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ARCHIVES OF SURGERY

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SPONTANEOUS RENAL AND URETERAL FISTULAS

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AND
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CLEVELAND

Renal and ureteral fistulas are uncommon, and when encountered usually are the result either of traumatic injuries or of surgical accidents. The spontaneous development of such urinary fistulas is so rare as to demand notation. Cases of nephrocolic, nephroperirenal, nephrochylous, ureterovesicoperitoneal and ureteroperiureteral fistulas have been studied at the Cleveland Clinic, all of which were the result of advanced pathologic processes which permitted the extravasation of urine into the adjacent organs and tissues.

Spontaneous renal and ureteral fistulas are unusual; their etiology is interesting, and they present difficult therapeutic problems; hence the following case reports.

REPORT OF CASES

CASE 1.—A woman, aged 21, was admitted to the Cleveland Clinic in January, 1929, because of pain in the left flank. For three years she had had recurrent attacks of "blind boils" over the anterior surface of the tibiae. The nodules had been sore and painful but had not ulcerated. Each crop had lasted for a period of one month, and then had subsided spontaneously.

Six weeks before coming to the clinic she had noticed pain in the left lumbar region radiating down the posterior aspect of the left thigh to the knee. At that time, the patient had been hospitalized for one week and had been told that she had "kidney disease." She complained of nocturia—four or six times—with urgency and burning on urination. She said that the urine was always cloudy and sometimes resembled milk. For two weeks the pain in the left lumbar region had become more intense, and the patient walked with a peculiar gait due to the inability to extend the left thigh completely. She had profuse night sweats, fever and occasional chills.

The physical examination revealed an asthenic, malnourished woman, apparently suffering from some chronic disease. Examination of the head, chest and heart gave essentially negative findings. The abdomen was symmetrical. Marked tenderness was present over the left costovertebral angle and flank. The muscles of the iliolumbar group were spastic and rigid, but extreme tenderness in this region precluded palpation of the kidney or detection of a perirenal mass. The pelvic organs were normal. The patient held the left thigh in a semiflexed position, and attempts to extend it fully caused pain in the left lumbar area. There were numerous small, firm, tender, erythematous nodules about 4 to 10 mm. in diameter scattered over the anterior surface of both tibiae. The temperature was 100 F.; the pulse rate, 126, and the blood pressure, 90 systolic and 58 diastolic.

From the Cleveland Clinic.

The red blood cells numbered 3,580,000; the white blood cells, 19,300, and the hemoglobin was 91 per cent. The blood sugar was 73 mg. and the urea 27 mg. per hundred cubic centimeters of blood. Serologic studies and repeated blood cultures gave negative findings.

Cystoscopic examination revealed a slight subacute cystitis. Both ureters were catheterized readily, and urine from the right kidney was normal, while a specimen from the left side was loaded with pus and Bacillus coli. All studies failed to show the presence of tuberculosis. In the differential renal function test the indigo carmine appeared on the right side in a concentrated form in five minutes, but none appeared on the left side in fifteen minutes.

A pyelogram of the left kidney showed a large, irregular, fragmented pelvis with extravasation of the pyelographic medium from the lower pole of the kidney

Fig. 1.—Pyelogram showing a large, irregular, fragmented pelvis with extravasation of sodium iodide from lower pole of left kidney into the colon, suggesting a nephrocolic fistula with coexisting perinephritic abscess.

into the colon (fig. 1). A diagnosis of a nephrocolic fistula with a coexisting perirenal abscess was made.

The patient was admitted to the hospital that evening, and because of her toxic condition she was given a transfusion of 500 cc. of blood. At operation on the following morning a large perinephritic abscess surrounding the left kidney was found, and 10 ounces of thick, foul-smelling pus escaped, cultures of which contained Bacillus coli. The kidney was enlarged and boggy. Most of the cortex had been destroyed by an active intrarenal infection. The patient's condition was too critical to permit a primary nephrectomy, so the perirenal abscess was drained.

Convalescence was uneventful until the ninth day after operation, when the temperature rose to 102 F. As this febrile reaction continued for eight days, it was thought that there might be a circumrenal collection of pus that was not draining.

Hence, the sinus tract was explored, but no pus was found. It appeared that the infection must be confined to the kidney. However, the patient continued to have fever, failed to gain in strength and did not improve sufficiently to undergo nephrectomy. She was discharged from the hospital, and a high vitamin diet, sun baths and absolute rest were advised. Improvement was very slow, and two years later she had a recurrence of pain in the left lumbar area, associated with fever, chills, pyuria and tenderness over the left kidney. At another hospital, a large perinephritic abscess was incised and drained.

During the next three months the patient improved considerably, and gained about 15 pounds (6.8 Kg.) in weight. For the third time she had a recurrence of pain over the left kidney associated with the classic signs and symptoms of a perirenal abscess. She was hospitalized, and Dr. Herrick explored the left kidney. He encountered a large perinephritic abscess which was drained. There was considerable difficulty in mobilizing the kidney, as it was firmly adherent to all perirenal structures. Extending from the renal pelvis to the splenic flexure of the colon was a firm band of fibrous tissue, which, on being severed, permitted a small amount of gas to escape from the bowel. Careful dissection showed this connective tissue strand to be a fistula which opened into both the colon and the renal pelvis. The opening into the bowel was closed by an inverted stitch and the kidney was removed.

In spite of the fact that all preoperative examinations had failed to show any evidence of acid-fast bacteria, microscopic studies of the kidney demonstrated areas of tuberculous ulceration. The patient improved considerably following the nephrectomy, but the last report stated that she was having urinary disturbance due to tuberculous cystitis and perhaps an involvement of the right kidney.

This case of nephrocolic fistula was the result of chronic renal tuberculosis which is the most common cause of nephro-intestinal fistula. In this case the renal tuberculosis had persisted for three years.

Case 2.—A man, 40 years of age, came to the clinic in December, 1930, complaining of "kidney trouble." He said that for the past sixteen years he had suffered recurrent attacks of severe colic in the left lumbar area, associated with fever, chills, pyuria and the passage of stones. Four years previously, the right testicle had become swollen, tender and slightly inflamed, and several small cutaneous sinuses had developed from which there had been a continuous purulent discharge. During the last four months there had been a constant, dull, aching pain in the left lumbar area associated with a sense of soreness and tenderness in the left costovertebral angle. He had nocturia about four times, always accompanied by a sense of incomplete evacuation of the bladder, urgency, tenesmus and burning on urination. Two weeks before he came to the clinic, swelling had appeared in the area of the left kidney, and since then he had been unable to extend the left thigh fully, as attempts to do so caused lumbar pains.

The patient had lost 32 pounds (14.5 Kg.) in weight during the past six months, in spite of a good appetite. He denied that he had had any pulmonary, cardiac or gastro-intestinal symptoms.

Physical examination showed an emaciated, asthenic man who walked with a peculiar limping gait, because of his inability to extend the left thigh fully. Examination of the throat, head, lungs and heart gave essentially negative findings. The abdomen was symmetrical, the muscles were atonic, and no tenderness or rigidity could be elicited. The temperature was 99.5 F.; the pulse rate was 120.

In the left costovertebral angle there was a fluctuant swelling. No definite mass could be outlined, but it seemed that a circumrenal collection of fluid was present. The skin overlying the tumefaction had a dusky red hue, indicating an inflammatory condition. No increase in local temperature could be detected.

The right epididymis was small, firm and indurated. When pressure was exerted on it, pus could be expressed from three cutaneous sinuses of the scrotum. Palpation of the prostate and seminal vesicles revealed no abnormality, and their secretions were normal according to microscopic evidence.

Laboratory reports were as follows: red blood cells, 4,380,000; white blood cells, 8,700, with a normal differential count; hemoglobin, 80 per cent; blood sugar, 70 mg. and urea 21 mg. per hundred cubic centimeters of blood. The Wassermann and Kahn reactions were negative in all antigens. The urine was loaded with

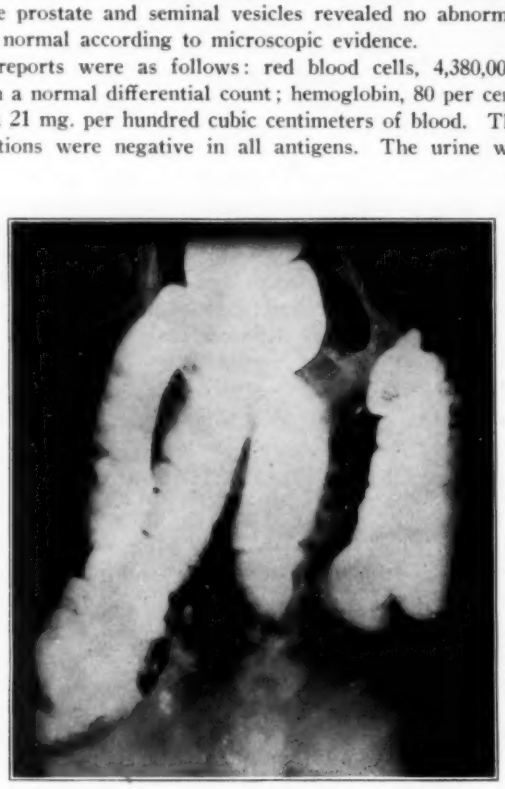

Fig. 2.—Pyelogram showing a nephrocolic fistula; barium is seen escaping from the splenic flexure of the colon and infiltrating the left perinephric region.

pus cells and contained numerous organisms, cultures of which revealed a mixed infection of Bacillus coli and staphylococci. All smears, cultures and animal inoculations of the sedimented urine were negative for tubercle bacillus. The renal function was good, as indicated by a 70 per cent excretion of the phenolsulphonphthalein dye administered by the intravenous method, and there was a normal range of specific gravity and concentrating power as shown by the Mosenthal test. Cystoscopic examination of the bladder indicated a slight inflammatory reaction of the mucosa but no definite signs of ulceration. In the differential kidney function test, the dye appeared from the right ureter, in a concentrated form, in four and one-half minutes; however, no dye was excreted by the left kidney in twenty minutes. Attempts were made to pass catheters and bougies up the left ureter, but an impassable obstruction was encountered about 1 cm. from the vesical orifice. Roentgenograms showed numerous areas of calcification in the left kidney which

suggested tuberculosis. Gastro-intestinal studies indicated that the stomach, duodenum and gallbladder were normal. An enema of barium sulphate filled the entire colon, and at the splenic flexure the barium escaped from the bowel and infiltrated the tissue around the left kidney (fig. 2).

The diagnosis suggested was nephrocolic fistula associated with perinephritic abscess.

Two days following the admission of the patient to the hospital, the fluctuant swelling in the left lumbar area ruptured spontaneously, a moderate amount of pus, undigested food particles and feces being discharged. It was hoped that by such drainage of the perinephritic abscess the patient's condition would improve; however, he continued to grow worse. Two days later, a second inflammatory swelling above the crest of the ileum in the left flank was incised, and a large quantity of foul pus was evacuated. Under a general medical regimen the patient improved slowly, but the failure of the urinary fistula to close and the persistent malodorous discharge, combined with a diseased, nonfunctioning left kidney indicated the necessity for operative intervention.

With the patient under spinal anesthesia, the left kidney was exposed, disclosing many dense perirenal adhesions. By careful dissection, it was possible to demonstrate a fistulous tract extending from the pelvis of the left kidney to the colon. The fistulous tract was excised, the opening in the colon was closed by two layers of inverted sutures, and nephrectomy was performed.

While the incision was being closed, the patient suffered acute shock. The respirations became rapid and shallow, and the pulse was weak, thready and almost imperceptible. The skin was cold and clammy. As an emergency measure, 500 cc. of whole blood and 300 cc. of 10 per cent solution of dextrose were given intravenously. The patient failed to respond to various measures of stimulation, and died about ten minutes after the incision had been closed.

At autopsy, multiple areas of ulceration were found at the splenic flexure of the colon and numerous small ulcers surrounded the ostium of the fistula. The parenchyma of the kidney was studded with multiple abscesses, but no gross or microscopic evidence of tuberculosis could be found. Examination of the right epididymis revealed a nonspecific inflammatory reaction.

Apparently the primary infection was intrarenal and was disseminated into the perirenal tissue, thus forming a perinephritic abscess. The abscess not only dissected its way into the colon but succeeded in forming a cutaneous sinus. Hence this case is typical for cutaneous, nephrocolic fistula.

Case 3.—A man, 29 years of age, a truck driver, entered the Cleveland Clinic in February, 1925, complaining of pain over the "left kidney." For three years, he had suffered from recurring attacks of boils, the last crop having appeared about two months previously. During the last seven weeks he had noticed a sharp pain in the left lumbar area, associated with slight fever in the afternoon. The urine was cloudy, and the patient had passed several blood clots, although he denied that he had had a frank hematuria. There was no history of frequency or burning on urination, and no chills or passage of urinary calculi. For three weeks he had been confined to bed because of progressive muscular weakness. A review of the pulmonary, circulatory and gastro-intestinal symptoms gave negative findings. The family history was irrelevant.

Physical examination revealed an asthenic man. His temperature was 100 F., the pulse rate, 100, and the respiratory rate, 20. Examination of the heart, lungs

and abdomen showed them to be normal grossly. In the left costovertebral angle there was a definite sense of tenderness associated with a suggestive muscular rigidity. However, there was no fluctuation and no increase in local temperature, and no tumor could be detected by palpation. Neither kidney was palpable.

Investigations in the laboratory showed that the red blood cells numbered 3,880,000, the white blood cells, 16,000, and the amount of hemoglobin was 80 per cent. The value for blood sugar was 80 mg. and for the urea 36 mg. per hundred cubic centimeters of blood. Both ureters were catheterized with ease. A specimen of urine from the left ureter was loaded with pus cells and contained numerous bacilli, none of which was acid-fast. The urine from the right kidney showed no abnormality. Pyelographic studies revealed areas of calcification on the mesial side

Fig. 3.—Pyelogram showing a nephroperirenal fistula with an irregular fistulous tract leading from the left renal pelvis into the perirenal tissue.

of the left kidney with an irregular fistulous tract leading from the pelvis of the kidney out into the perirenal tissue (fig. 3). Evidently the pyelographic medium escaped from the kidney out into a coexisting perinephritic abscess.

The presence of pyonephrosis with an associated perinephritic infection having been demonstrated, it was thought advisable to remove the left kidney. At operation a large perirenal abscess was found to surround the left kidney completely and to communicate by means of a fistula with the renal pelvis. The kidney was enlarged, the cortex was soft, and numerous abscesses were present. The renal pedicle was ligated, the kidney was removed, and the perinephritic abscess was drained. A careful microscopic examination failed to reveal any evidence of renal tuberculosis, and cultures from the perirenal and kidney abscesses were negative for acid-fast bacilli. Convalescence was satisfactory, and the patient was apparently well at the last report.

This case of nephroperirenal fistula apparently represents the so-called metastatic kidney infection, the primary focus being probably the "cutaneous boils." The existing pyuria was caused by the rupture of the perinephritic abscess into the renal pelvis.

CASE 4.—A woman, aged 46, entered the Cleveland Clinic in April, 1925, complaining of pain in the left lumbar area. Eight months previously, she had suffiered from influenza which she said had "settled in her kidneys." Since then she had suffered from recurring attacks of colic which always was localized in the left renal area. The pains were initiated by working, the attacks lasting for one or two days, and almost always morphine was required for relief. At times the

Fig. 4.—Pyelogram showing a ureteroperiureteral fistula; there is a duplex kidney pelvis with irregular nodular calices. At the level of the left sacro-iliac joint, a loop of the ureter deviates toward the vertebral column, and at this point the pyelographic medium escaped from the ureter and infiltrated the periureteral tissues.

patient suffered from urgency, frequency and burning on urination. Pus, albumin and occasional red blood cells had been found in the urine. She had had no frank hematuria and had never passed urinary calculi.

During the three weeks prior to her admission to the hospital, the attacks of "kidney pain" had increased in severity and frequency, being associated with nausea, vomiting, chills and mild fever each afternoon. She had no desire for food, and had lost 50 pounds (22.7 Kg.) in weight during eight months.

Physical examination revealed that the patient was greatly emaciated. There was no evidence of acute pain, and all mental processes were normal. Examination of the heart and lungs gave negative findings. On inspection, a definite fulness was found just beneath the left costovertebral angle with a decreased excur-

sion of the diaphragm on that side. The left iliolumbar muscles were very spastic, and the entire area was very tender. No definite mass could be detected because extreme tenderness precluded a careful examination. The skin was normal, and no evidence of fluctuation or abscess formation could be detected. Her temperature was 101 F., and the pulse rate was 110.

Red blood cells numbered 3,470,000, the white blood cells, 18,300, and the hemoglobin value was 65 per cent. The blood sugar was 87 mg. and the urea 39 mg. per hundred cubic centimeters of blood. A catheterized specimen of urine from the left kidney was loaded with pus and contained many nonacid-fast bacilli, while the urine from the right kidney was normal. The combined renal function according to the intravenous phenolsulphonphthalein test was 55 per cent in two hours.

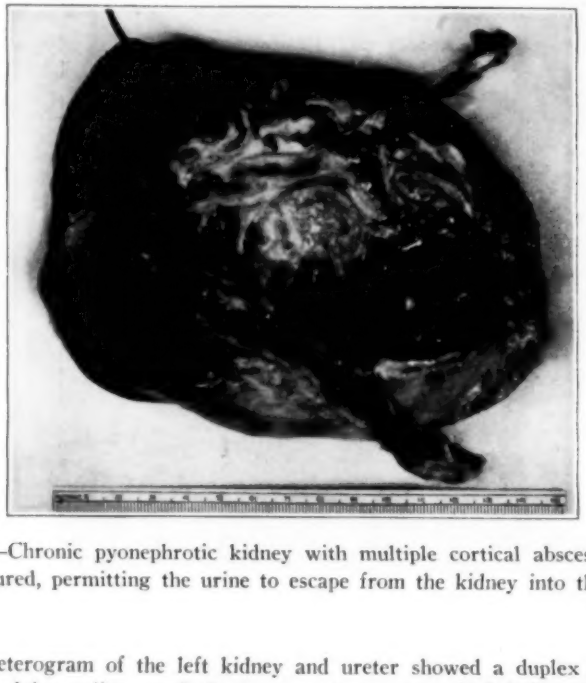

Fig. 5.—Chronic pyonephrotic kidney with multiple cortical abscesses, one of which ruptured, permitting the urine to escape from the kidney into the perirenal space.

A pyelo-ureterogram of the left kidney and ureter showed a duplex pelvis with irregular nodular calices, and the two ureters were united before entering the bladder. At the level of the left sacro-iliac joint, a loop of ureter deviated toward the vertebral column, and at this point the pyelographic medium escaped from the ureter and infiltrated the periureteral tissue (fig. 4).

The foregoing findings suggested the preoperative diagnosis of duplex kidney with pyonephrosis associated with ureteral-periureteral fistula.

On exposure of the left kidney, a large perinephritic abscess was found from which 10 ounces of pus escaped (fig. 5). The kidney was about four times the normal size, and the cortex was soft and riddled with multiple abscesses. Three hundred cubic centimeters of thick pus was aspirated from the renal pelvis in order to expedite a nephrectomy. The perirenal abscess had extended along the ureter, and at the level of the left sacro-iliac joint a large periureteral abscess was found.

This abscess cavity contained pyelographic solution, but the fistulous tract could not be isolated on account of the pronounced inflammatory reaction of all the perireteral tissue. However, the ureteral sinus was visualized by the pyelo-ureterogram, and the extravasation of the urographic fluid clearly indicated the presence of a ureteral fistula.

Convalescence was slow, but when the patient was last heard from she was enjoying good health.

This case illustrates the complications that may accompany pyonephrosis. The kidney was evidently the primary source of infection, and the inflammatory process extended to the perinephrium, producing an abscess. The perirenal pus dissected its way down the outside of the ureter, and at the junction of the middle and lower thirds it eroded the ureteral walls and evacuated its contents into the ureter, thus accounting for the coexisting pyuria.

Case 5.—A laborer, 44 years of age, entered the Cleveland Clinic in October, 1930, complaining of "bladder trouble." He had been well until eight months before, when he had noticed a total hematuria which persisted for two weeks and then subsided spontaneously. Since then, the urine always had been cloudy. He had suffered from frequent urination, every two hours, associated with burning in the urethra. He denied lumbar pains and had never passed urinary stones. His general health had been good, but he had lost 10 pounds (4.5 Kg.) in weight during the last six weeks owing to voluntary restriction of diet. There was no familial history of tuberculosis.

The physical examination revealed that the patient was well nourished. His temperature was 98.6 F., the pulse rate was 76, and the blood pressure 136 systolic and 84 diastolic. His weight was 175 pounds (79.4 Kg.) (normal weight, 185 pounds [83.9 Kg.]). The only physical abnormality noted was slight prostatic enlargement. The gland was soft and boggy, and the secretion contained from 30 to 50 pus cells per high power field.

Cystoscopic examination revealed a chronic inflammation of the entire mucosa of the bladder, with localized areas of ulceration. The right ureteral orifice was surrounded with areas of bullous edema. Several obstructions suggesting multiple ureteral strictures were encountered in passing a spiral tipped catheter up to the pelvis of the right kidney. About 50 cc. of thick, cloudy urine was aspirated from the pelvis of the right kidney. The left kidney and ureter were normal. The indigo carmine dye was excreted from the right kidney in dilute form, in ten minutes, and from the left in concentrated form, in five minutes. Sixty per cent of the phenolsulphonphthalein dye was excreted in two hours.

Pyelographic studies indicated a chronic inflammation of the pelvis of the right kidney with cicatricial contraction of the lower calices and multiple strictures of the ureter. There were two definitely circumscribed shadows involving the lower end of the right ureter, which suggested the presence of diverticula. The pelvis of the kidney was irregular, and several small calcified areas were seen in the renal cortex. Dr. Nichols made the roentgenographic diagnosis of renal tuberculosis with a coexisting cystitis. However, catheterized specimens of urine were examined repeatedly before the presence of acid-fast organisms could actually be demonstrated.

On Nov. 16, 1928, nephrectomy was performed in a local hospital. Convalescence was uneventful, except that the wound refused to heal. Four weeks later the patient was admitted to the Cleveland Clinic. The temperature ranged from 98.6 to 103 F. The patient perspired freely, and complained of urgency and burning on urination. Approximately one half of his urine escaped through the right lumbar sinus in spite of the fact that the right kidney had been removed. Evidently there was a urinary fistula connecting the lumbar incision with the bladder. Cystoscopic examination failed to show such a fistula, but the bladder mucosa was so intensely inflamed and covered with punched-out ulcers that the examination was unsatisfactory.

During a long period of hospitalization, numerous therapeutic measures were employed in an attempt to control the hyperpyrexia and the cystitis and to facilitate closure of the incision, but all to no avail. The patient complained of tenderness in the right lower abdominal quadrant; the overlying muscles were spastic. He was very somnolent. His neck was stiff, and there was a bilateral ptosis of both eyelids. On March 30, 1929, seventy-five days after admission, he died from generalized miliary tuberculosis with signs of tuberculous meningitis.

The diagnosis of generalized miliary tuberculosis was confirmed at necropsy. The bladder was small and inflamed, and the mucous membrane was necrotic. The dome of the bladder on the right side was sacculated and communicated with a sinus tract leading to a large abscess located in the lower right abdominal cavity. The abscess contained a large amount of purulent material having an ammonical odor of decomposing urine. A second sinus connected the right ureter with the abscess cavity, and a third sinus joined the abscess with the lumbar wound. Thus the urine secreted by the left kidney escaped from the bladder by means of the vesicoperitoneal fistula or by going up the stump of the right ureter and escaping through the ureteroperitoneal fistula. It is true that the lumbar incision was caused by the previous operations, but the pelvic and the ureteral and vesical fistulas were of spontaneous origin. Examination of the right ureter revealed that the opening of the fistulous tract had been distal to the point of ligation during the previous nephrectomy. The entire pelvis and extravesical structures were involved in an extensive tuberculous inflammation, and the degenerative changes had so involved the wall of the ureter and bladder that they finally perforated.

The following case of urinary chylous fistula has been reported by Lower and Belcher of the Cleveland Clinic.[1] The case is summarized here in order to complete our present study.

CASE 6.—A Chinese cook came to the Cleveland Clinic in July, 1923, because of "bladder trouble." Five months previously he first had noticed that his urine was cloudy; at times it was so viscid and hazy that it resembled milk. There was no burning on urination, no urgency and no gross hematuria. During this period he had lost 10 pounds in weight, although his appetite was good. He had no pulmonary, cardiac or gastro-intestinal symptoms.

He was a native of China, but had lived in the northern part of the United States for ten years.

No gross abnormalities were noted on physical examination.

The blood count was normal, and no parasites were found, although repeated examinations were made at all hours of the day and night. Numerous blood

1. Lower, W. E., and Belcher, G. W.: Chyluria: With a Report of a Case Treated with Neosalvarsan, Surg., Gynec. & Obst. **39**:147, 1924.

Wassermann and Kahn reactions were negative in all antigens. The blood sugar was 85 mg. and the blood urea 33 mg. per hundred cubic centimeters of blood.

The bladder was normal on cystoscopic examination. The urine collected from the right kidney was clear, while that obtained from the left side resembled cream. A chemical analysis of the urine by the Babcock method showed 1 per cent fat. The fat content was so high that its presence could be demonstrated grossly by its leaving a grease spot on absorbent paper. Repeated pyelographic studies during and between attacks failed to demonstrate any fistulous communication between the kidney and the lymphatic system.

The patient was given a course of neoarsphenamine, and the chyluria immediately subsided. However, he had severe recurrent attacks, and each time the urine gave a positive test for fat. The most unusual aspect of the case was the specificity to neoarsphenamine even though no Filaria were found.

While no fistulous tract was actually demonstrated, it seems logical to believe that there was an artificial communication between the lymphatics and the kidney which permitted the extravasation of the lymph into the urinary tract. Hampton [2] likened "the deep lymphatics of the kidneys to the cerebral arteries, as the points of lowest resistance in their respective systems and assumes that the lymphatic obstruction results in a leakage causing a chyluria just as hypertension often results in leakage which causes apoplexy."

From a study of the foregoing cases it is evident that renal and ureteral fistulas may result from a number of pathologic conditions; therefore, each type of fistula will be discussed separately and clinical examples will be cited from the literature.

RENAL FISTULA

Spontaneous perforation of the kidney accompanied by fistulous communication with adjacent organs or tissues usually is the result of advanced renal disease such as pyonephrosis, nephrolithiasis, neoplasm, tuberculosis or the various suppurative nephritides. However, the kidney may become damaged secondarily as a result of a ureteral obstruction with its coexisting hydronephrosis, or a perinephritic abscess may rupture into the renal pelvis, or a neoplasm of the bowel may extend and involve the kidney.

Renal fistulas may be classified according to etiology or according to their anatomic relationships, the latter being the most satisfactory classification, as the location of the fistula and organs with which it communicates are designated. We have followed the anatomic classification and have searched the literature for cases which represent the various etiologic processes.

2. Hampton, H. H.: Non-Parasitic Haemotochyluria, Bull. Johns Hopkins Hosp. **31**:20, 1920.

The fistulous tract may originate from the renal parenchyma or from the pelvis and may open into the stomach, intestines, lungs, peritoneal cavity or into the circumrenal tissue, from whence the urine may work its way out through the lumbar muscles and perforate the skin. The sinus tract usually is direct, but may be long and sinuous, and opens into the kidney by one ostium; however, there are a few exceptions to this rule.

The material discharged from the fistula depends on the pathologic status of the kidneys. If a calculous pyonephrosis is present, then pus and stones may escape; if the renal tissue can function, then urine may be detected in the discharge. If the fistula is caused by a rupture of a long-standing hydronephrosis with atrophy of the secreting tissue, the examination of the escaping fluid for urea, creatine and urinary phosphates is negative. Failure to recognize this point may result in an incorrect diagnosis. In tuberculous pyonephrosis the discharge may contain caseous plugs of necrotic material, and the ostium of the sinus has the appearance of unhealthy granulating tissue. If the kidney is actively secreting, incrustations of urates and phosphates may be detected in the walls and lumen of the sinus. When the sinus communicates with the gastro-intestinal tract, stones, pus and urine may be found in the vomitus and feces. Food particles may also be expelled from the sinus or may be found in the bladder.

Renogastric Fistula.—Authentic examples of communications between the left kidney and the stomach are rare indeed; however, several probable cases have been reported, the diagnosis having been made on account of the vomiting of stones, pus and urine.

Morris[3] treated a woman, who for eleven months excreted pus in the urine and feces. Five months later a tender swelling appeared in the left loin, which finally ruptured, a pint of pus being discharged. Multiple cutaneous fistulas developed, one over the twelfth rib and three over the left sacro-iliac region, all of which discharged pus continuously. The cutaneous sinuses were dissected out, and all led to the kidney. No evidence of osteomyelitis of the ribs or vertebrae was found. The next day the patient had complete anuria with six loose bowel movements which contained pus. At autopsy, the left kidney was found to be enlarged and contained multiple abscesses. Opening into one of these caseous and suppurating cavities of the kidney was a fistulous tract which communicated with the superficial abscess of the loin. A second sinus opened into the left margin of the greater

3. Morris, Henry: Surgical Diseases of Kidney and Ureter, London, Cassell & Co., 1901, vols. 1 and 2.

curvature of the stomach, and by exerting pressure on the renal pelvis, pus could be expressed into the gastric cavity. No statement was made in the case report as to whether or not tuberculosis was present.

Morris also mentioned a case reported in 1678 of a woman 21 years of age, who after an attack of intense renal colic with anuria, vomited coffee-colored gravel weighing half an ounce. Later, she passed urinary calculi by urethra, mouth and rectum. He referred to another patient suffering from urinary colic from whom a stone was removed from the ureteral orifice by cystoscopic manipulations. Subsequently the symptoms recurred, at which time the patient vomited, voided and defecated urinary stones.

Rayer [4] reported a gastronephric fistula occurring as the result of tuberculosis of the kidney, which is the most common cause of this type of fistula. Chadwick [5] cited a case in which particles of undigested food were withdrawn from the bladder and presented this as evidence of a renogastric sinus, believing that the undigested food must, of necessity, have escaped from the stomach, otherwise it would have been partially digested. Hilgenreiner [6] described a case of his own and cited those of Melion,[7] Marquezy,[8] Murchison,[9] Richter [10] and Kuster [11] as probable cases of fistulas occurring between the kidney and the stomach. To those cases may be added those of Riverius,[12] Fernelius [13] and Rosenbach.[14] In many of these reports the pathologic data are so indefinite that no statement as to the incidence of tuberculosis in this type of lesion can be made.

The presence of pus, urine or stones in the vomitus does not establish the diagnosis of nephrogastric fistula, as nephroduodenal or nephrocolic sinuses may produce the same symptoms. The appearance of food particles in the urine usually indicates an enterovesical sinus. Gastrourinary fistulas have been suspected when the vomitus contained urine, but several interesting cases have been studied in which the trouble has been found to be due to psychopathic malingering.

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4. Rayer, cited by Morris.[3]
5. Chadwick, J. R., cited by Weiser.[30]
6. Hilgenreiner, cited by Weiser.[30]
7. Melion, cited by Weiser.[30]
8. Marquezy, T. P.: Une observation de fistula néphrogastrique, Thèse de Paris, 1856, no. 28, v. 593.
9. Murchison, cited by Weiser.[30]
10. Richter, cited by Weiser.[30]
11. Kuster, cited by Weiser.[30]
12. Riverius: Observationes medicæ et curationes insignes, quibus accesserunt observations ab aliis communicatæ, London, M. Flesher, 1646.
13. Fernelius, cited by Morris.[3]
14. Rosenbach, O.: Ein Fall von Pyelitis calculosa mit Perforation in das Colon ascendens, Inaug. Dissert., Breslau, 1873.

Nephroduodenal Fistula.—Nephroduodenal fistulas are much less common than those of the gastronephric type, only three cases of the former having been reported in the literature. Rayer (cited by Chazet [15]) described a case observed by Campaignac, that of a tailoress, 45 years of age, who had noticed a swelling of the right flank. At times she suffered from intense nausea, and had vomited large amounts of foul, acrid, urine-like material. During the attacks of vomiting a spontaneous decrease in the size of the lumbar tumor occurred. Necropsy revealed a communication between the collapsed pyonephrotic kidney and the first part of the duodenum. Also two small sinus tracts joined the perinephritic abscess with the renal pelvis.

Bang [16] treated a patient who had renal and ureteral tuberculosis. The indurated ureter was partially occluded by the inflammatory process and produced a hydronephrosis which caused a fusion and finally an abnormal communication between the kidney and the duodenum. Fernelius presented an incomplete report of a duodenal-kidney fistula which he observed in 1646.

Nephrocolic Fistula.—Inflammatory lesions may produce communications between the kidney and any part of the large intestine. As a general rule, pyonephrosis, tuberculosis and perinephritic abscesses are the most common offenders.

Rayer described a case of renal tuberculosis in which a sinus tract joined the kidney and colon; two cutaneous sinuses also opened into the loin, forming a renocolic cutaneous fistula. Rayer referred to another example of tuberculous necrosis of the kidney and colon in which there was a patulous communication. D'Allaines and Rouffiac [17] treated a man who had a large tumor in the right part of the hypochondrium. An exploratory laparotomy revealed a large perinephritic abscess surrounding a kidney with caseous degeneration, the abscess having ruptured into both the kidney and the colon.

Ogle [18] observed a patient with a large perinephritic abscess which ruptured into the colon, causing a purulent diarrhea with expulsion of urinary calculi; the diagnosis was confirmed at autopsy. Morris mentioned the case of Ammundale,[19] who incised a large psoas abscess

15. Rayer, cited by Chazet, Gaston: Contribution à l'étude de la tuberculose rénale avec fistules, Thèse de Paris, 1899-1900, no. 374.

16. Bang, B. F. L.: Primaer tuberculose i urogenitalorganerne; kommunikation immellem duodenum og den højere ureter, Hosp.-Tid. Københ. **1**:561, 1874.

17. d'Allaines and Rouffiac: Fistule pérenéphrocolique avec lésions inflammataires du rein, Bull. Soc. anat. de Paris **93**:324, 1923.

18. Ogle, J. W., cited by Weiser.[30]

19. Ammundale, cited by Morris.[3]

which was pointing in the lumbar area. Fifteen days later urine and feces were discharged from the incision. The psoas abscess was secondary to the calculous pyonephrosis.

External abscesses, arising from perforation of the kidney substance by a small stone, may open into the colon and discharge pus, urine and calculi into the intestinal tract, as observed by Rosenbach.

Dittrich [20] saw an appendicorenal fistula resulting from a ruptured appendix with a subsequent perinephritic abscess which perforated the kidney, thus forming a patulous tract between the kidney and the appendix. Cruveilhier [21] referred to a woman who was reported to have had a "septic death." At autopsy, both kidneys were found to be in the true pelvis and fused together behind the rectum. As a result of pyonephrosis, the infection had spread to the perirenal tissue and had formed an abscess which perforated the rectum, forming a renorectal fistula.

To this series may be added the two cases of nephrocolic fistula studied at the Cleveland Clinic. In one case the fistula between the kidney and colon communicated with a second sinus, which extended through the lumbar muscles, permitting undigested food to escape from the cutaneous opening.

Renopulmonary Fistula.—Communications between the kidney and the lungs, as suggested by the expectoration of pus, urine and urinary calculi, usually signify advanced renal disease. This is illustrated by tuberculosis of the kidney with its resulting perinephritis, which may lead to a perirenal abscess, the infection then spreading along the surface of the liver forming a subdiaphragmatic abscess which may perforate the diaphragm, producing an empyema, with final escape of the pus by erosion into one of the bronchi.

Morris referred to the case cited by Rayer, that of a locksmith, 39 years of age, who eighteen years before had had a stone removed from the bladder. For eight years he had suffered severe pains in the left lumbar area associated with sudden pyuria. The pains were sometimes referred along the course of the ureter. On two occasions a profuse purulent expectoration occurred with a simultaneous disappearance of the pyuria. Autopsy disclosed a communication between the bronchus of the lower left lobe of the lung and the kidney. The fistulous tract had extended through a dilated calyx, penetrating the diaphragm and opening into a bronchus; several small phosphatic concretions were lodged in the lumen of the sinus. The kidney was practically destroyed by tuberculous caseation.

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20. Dittrich, cited by Weiser.[30]
21. Cruveilhier, cited by Morris.[30]

Roubier [22] and Pillet [23] each encountered a case of tuberculous nephritis in which erosion had occurred through the renal cortex, and the resulting perinephritic abscess had slowly bored its way through the diaphragm and had finally emptied into a bronchus. Hortels [24] reported a cold psoas abscess which evacuated itself by emptying into the renal pelvis, producing pyuria, and also opened into a bronchus causing a foul purulent expectoration. Gordon [25] described a case in which there was tuberculous infection of the kidney and a perinephritic abscess which ruptured into the lungs, associated with multiple ulcerated areas in the duodenum.

Van der Speck [26] treated a patient who coughed up thin purulent material containing urinary salts. When the patient expectorated a large quantity of this material, there was a simultaneous decrease in the size of the lumbar tumor. Urologic examinations indicated that this was a case of congenital dystrophy of the left kidney with a coexisting perinephritic abscess which had perforated the diaphragm and was draining into a bronchus.

Von Deesten [27] described a patient who coughed up a copious amount of foul purulent sputum whenever he lay down. Physical findings and roentgenograms of the chest were normal. A diagnosis of perirenal abscess was made. When the abscess was incised and drained, all pulmonary symptoms immediately subsided. Von Deesten believed this to be a case of renopulmonary fistula although the sinus tract was not actually demonstrated.

Spontaneous rupture of a large hydronephrotic kidney into the perirenal tissues may cause an empyema which perforates into a bronchus; in such a case Kondratovich [28] found pus and urine in the sputum. In 1910, McDwean [29] collected five cases of direct renopulmonary fistula, the majority being caused by tuberculous infection of the kidney.

Renoperitoneal Fistula.—Nontraumatic perforation of the kidney with extravasation of urine, pus and stones into the peritoneal cavity is rare, only six cases having been reported.

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22. Roubier, C.: Tuberculous Kidney-Lung Fistula, J. d'urol **13**:195, 1922.
23. Pillet, cited by Weiser.[30]
24. Hortels, cited by Chazet.[15]
25. Gordon, S.: Case of Reno-Pulmonary Fistula with Ulceration of Duodenum, Dublin Quart. J. M. Sc. **41**:90, 1866.
26. Van der Speck, H.: Uroptysis, Nederl. tijdschr. v. geneesk. **2**:1855, 1923.
27. von Deesten, H. T.: Recumbent Posture Cough with Purulent Expectoration; An Indicative Symptom in a Case of Perinephritic Abscess which had Ruptured into a Bronchus, J. A. M. A. **88**:98 (Jan. 8) 1927.
28. Kondratovich, K.: Pyohydronephrosis with Elimination of Urine Through the Bronchus, Med. Obozr., Moscow **51**:591, 1899.
29. McDwean, cited by Weiser.[30]

Weiser [30] mentioned a case of calculous pyonephrosis which ruptured into the peritoneal cavity, producing localized peritonitis, splenic and subdiaphragmatic abscesses and empyema due to the infectious process spreading by way of the lymphatics to the pleural cavity. The infection also invaded the circumrenal tissue, producing a perinephric collection of pus.

Rupture of a kidney in which there has been long-standing hydronephrosis, and in which the walls of the renal pelvis have been stretched out to paper thinness, accounts for most urinary ascites. In the case reported by Bennett,[31] a ureteral stone had produced a progressive hydronephrosis which ruptured into the abdominal cavity. An immediate operation with aspiration of the urinary ascites and a repair of the ruptured kidney resulted in rapid convalescence. Taylor [32] referred to the case of a girl, 15 years of age, in whom a large cystic tumor extended from the left costal margin to the crest of the ilium. One night the cyst ruptured spontaneously, and on the next morning an exploratory laparotomy was performed, at which time urine was encountered in the peritoneal cavity. Since it was impossible to locate the artificial opening in the kidney, the hydronephrotic sac was sutured to the anterior abdominal wall, thus forming an external urinary fistula which permitted the continuous drainage of urine. On several occasions the ostium of the fistula closed, and immediately the patient had a recurrence of the kidney pain; as soon as the sinus tract was opened, the pain was relieved.

Turner [33] performed a laparotomy because of a ruptured hydronephrosis of a single kidney and found peritonitis due to urinary irritation and infection. Later, peritoneal abscesses developed, and the patient died. Bazy [34] has pointed out that if the urine is sterile, the resulting chronic peritonitis usually responds to surgical treatment, but if the escaping urine is infected, the patient rapidly succumbs to a fulminating peritoneal infection.

The diagnosis of urinary peritonitis is difficult, as attested by the following reports. Schreuder [35] performed an operation for appendi-

30. Weiser, A.: Die nichttraumatischen Perforation und Fisteln des Harntrakes, Ztschr. f. urol. Chir. **28**:120, 1929.

31. Bennett, cited by Morris.[3]

32. Taylor, J. W.: Acute Hydronephrosis or Dilatation of the Pelvis of the Left Kidney: Rupture of Sac and Extravasation of Urine into Peritoneal Cavity; Operative Recovery with Rupture into Peritoneal Cavity, Lancet **2**:589, 1884.

33. Turner, Phillip: Hydronephrosis of Single Kidney: Spontaneous Rupture into the Peritoneal Cavity, Proc. Roy. Soc. Med. **16**:24, 1923.

34. Bazy, cited by LeComte.[42]

35. Schreuder, O.: Spontaneous Rupture of Hydronephrosis, Deutsche Ztschr. f. Chir. **191**:100, 1925.

citis and encountered free urine in the abdominal cavity due to a kidney ruptured by pyonephrosis. Tschugajeff [36] explored the abdomen for a cryptogenic peritonitis and was surprised to find a ruptured kidney with infected urine draining into the peritoneal cavity.

Renoperirenal Fistula.—The simplest and most common renal fistula is that caused by a rupture of the kidney which permits the extravasation of urine into the circumrenal tissue with the formation of perirenal and psoas abscesses. There are numerous pathologic conditions which might cause spontaneous rupture of the kidney.

It is not uncommon to find a perinephritic abscess which empties into the renal pelvis, causing intense pyuria. Alglave [37] encountered a case of tuberculous spondylolisthesis with an associated psoas abscess; the pus finally perforated the kidney, causing a sudden copious pyuria. In Weiser's patient the cold abscess developed from the dorsal vertebra and not only did it rupture into the kidney pelvis but the infection spread under the diaphragm, the bacteria reaching the pleural cavity by lymphatic dissemination resulting in empyema. Maudet [38] described a case in which there was a tuberculous kidney with multiple areas of caseation which finally perforated the kidney, and the infected urine infiltrated the perirenal tissues, causing a large perinephritic abscess. When the perinephritis is the result of a renal fistula, the pus usually collects posterior to the kidney. However, the fluid may be anterior to the kidney and push the peritoneum forward, forming a cystic mass which can be palpated through the anterior abdominal wall, and often is mistaken for an intra-abdominal tumor. Morris performed an autopsy on a patient who had died of scrofulous pyonephrosis and cystitis, and found a large retroperitoneal abscess which had dissected its way downward from the left kidney to the brim of the pelvis and the anterior crest of the ilium.

Vanwerts [39] cited the case of a woman, 25 years of age, who had undergone an operation for appendicitis, at which time a perityphlic abscess had been found and drained. Five days later urine was discharged from the wound. He felt that the perityphlic infection spread through the retroperitoneal tissue, causing a psoas abscess which ruptured into the renal pelvis. By the use of indwelling ureteral catheters, the patient made a satisfactory recovery. However, there is no certainty that the abscess did not perforate the ureter instead of the renal pelvis.

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36. Tschugajeff, cited by Weiser.[30]
37. Alglave, cited by Weiser.[30]
38. Maudet, cited by Chazet.[15]
39. Vanwerts, cited by Weiser.[30]

To this series of cases may be added our case 3 in which the perinephritic abscess eroded through the thin pelvic wall, giving rise to pyuria.

Spontaneous rupture of a hydronephrotic kidney is another cause of urinary extravasation into the circumrenal tissue. A persistent ureteral obstruction may produce a progressive hydrostatic dilatation of the renal pelvis to such a degree that it may rupture. Van Saar [40] observed an unusual case in which spontaneous perforation of the hydronephrotic kidney occurred twice. The first rupture of the renal pelvis permitted the urine to escape into the perirenal tissue, and under conservative treatment the patient made an uneventful recovery. Eighteen months later a severe pain in the area of the left kidney announced the second rupture. During the process of nephrectomy, urine was encountered in the perirenal tissue, and the rent in the pelvic wall was demonstrated in the removed specimen. Van Saar believed that the spontaneous healing of the renal pelvis following the first rupture was due to the sudden release of the intrapelvic pressure which permitted coaptation of the pelvic walls with consequent healing. The kidney tissue was functioning so slowly that the reparation was complete before sufficient fluid had collected to cause another rupture. The extravasated fluid consisted chiefly of water and sodium chloride; hence it was readily absorbed. Had the urine been infected, a perinephric abscess probably would have resulted. An incarcerated calculus at the ureteropelvic junction may lead to a ruptured hydronephrosis and permit the urine to escape into the perirenal tissue. Mathe and Oviedo [41] observed such a case in which, following a pelviolithotomy and plastic repair of the perforation, the patient made an uneventful recovery. When the extravasated urine is mixed with sufficient blood to form a perirenal hematoma, as in LeComte's [42] case, the rent in the hydronephrotic kidney usually involves both the pelvic wall and the cortex. Wood [43] performed an exploratory laparotomy for an anticipated acute peritonitis and discovered a large cystic tumor surrounding the right kidney. He incised the mass, encountered free urine, and found a hydronephrotic kidney with a small opening into the pelvis whence urine had escaped.

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40. Van Saar, G.: Ueber Hydronephrosenruptur und den dabei auftretenden Symptomenkomplex, Beitr. z. klin. Chir. **64**:316, 1909.

41. Mathe, C. P., and Oviedo, G. F.: Spontaneous Rupture of Hydronephrotic Sax, Secondary to Ureteral Stone, California & West. Med. **26**:790, 1927.

42. LeComte, R. M.: Spontaneous Rupture of Hydronephrosis, J. Urol **15**:517, 1920.

43. Wood, W. F.: Spontaneous Rupture of Hydronephrosis, Brit. J. Surg. **10**:574, 1923.

Renal calculi generally are associated with chronic urinary stasis, infection and pressure necrosis, all predisposing to spontaneous perforation of the cortex of the kidney. If the stones are small, they may escape into the perinephric space and then migrate to the lower lumbar area, where they cause a psoas abscess, such as that observed by Pachkis.[44] However, a solitary, large dendritic stone may penetrate the cortical tissue and still remain lodged in the renal pelvis. Everett[45] performed a pelviolithotomy for such a condition, but the patient died from the absorption of infected urine.

Nephrolithiasis is usually associated with functioning renal tissue, and when the stone pierces the medulla and cortex, the extravasated urine may infiltrate all the perinephric tissue and produce pronounced constitutional reactions. Sometimes the escaping urine follows the course of the ureter, and a periureteral abscess may result, as was described by Turner.[46] Occasionally the urine dissects its way along the fascial and muscular planes, causing an inflammatory edema in distant areas. Heniline treated a patient with a tender inflammatory swelling of the scrotum, perineum and lower portion of the abdomen. Multiple incisions of the edematous area permitted the escape of foul material which resembled urine. At autopsy it was found that the upper calyx of the kidney had been perforated by a small stone, forming a nephroperirenal fistula. An unusual case of urinary extravasation was seen by Delzell and Harrah.[47] Their patient had a diffuse cellulitis of the penis, scrotum and thighs. The anterior abdominal wall and the lumbar muscles were involved in an extensive inflammatory reaction. The peritoneal cavity was filled with multiple abscesses, and the liver was pushed downward by a subdiaphragmatic collection of pus. The pleural cavity contained a localized empyema and was covered by a plastic exudate. This generalized systemic infection resulted from the spontaneous perforation of a pyonephrotic kidney.

A fourth cause for urinary extravasation into the circumrenal tissue is the spontaneous perforation of the renal cortex. Many cases of idiopathic rupture of the parenchyma of the kidney have been reported, but extravasation of urine was present in only a small percentage of these cases. The cortex of the kidney seems to be more friable than the pelvis and ruptures more frequently, but unless the

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44. Pachkis, R., cited by Weiser.[30]

45. Everett, William: Perforation of Kidney by a Large Branched Calculus, Brit. M. J. **1**:17, 1925.

46. Turner, Alfred C.: Unusual Case of Urinary Fistula, Lancet **2**:220, 1929.

47. Delzell, W. R., and Harrah, Frank W.: Eleven Cases of Ruptured Kidney, J. Urol. **18**:131, 1928.

rent extends through all the cortical tissue and opens into the pelvis, or unless the entire kidney is split open, only perirenal hematoma is formed and no urine escapes.

Fox [48] described the case of a man, who on tripping and attempting to prevent falling, had performed some unusual muscular movements which caused pain in the right lumbar area. Exploration revealed a complete transverse rupture of the kidney with a perirenal hematoma mixed with urine. Many authorities are of the opinion that a kidney must be weakened by disease before it can be ruptured by muscular exertion.

In Brown's [49] case, two stones obstructed the ureter producing a pressure atrophy of the renal cortex which finally resulted in a rupture of the parenchyma of the kidney with liberation of urine into the perirenal tissue.

Rupture or perforation of the kidney substance may occur as the result of renal neoplasms, such as hypernephromas, carcinomas, embryomas or sarcomas. As the tumor growth is rapid, degenerative changes often result in necrosis of the renal tissue with the formation of urinary sinuses. Tuffier [50] described a case of renal carcinoma, and Hartmanns [51] cited the case of an unidentified neoplasm, both of which caused a spontaneous rupture of the cortex with urinary extravasation into the perinephrium.

Renocutaneous Fistula.—Renal fistulas which open exteriorly usually point in the loin, flank or groin. This type has received considerable attention in medical literature.

Tuberculosis of the kidney is the most frequent cause of cutaneous urinary fistula and represents advanced renal lesions. Barney and Mixter [52] treated a patient with renal tuberculosis complicated by an inguinorenal fistula, nephrolithiasis and transverse myelitis of the spinal cord.

Schwartz [53] encountered a tuberculous perinephritic abscess which not only produced pyuria by rupturing into the kidney but also caused

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48. Fox, O. E.: Ruptured Kidney Due to Muscular Action, Atlantic M. J. **31**:183, 1927.

49. Brown, R.: Spontaneous Rupture of Renal Pelvis, Surg. Clin. North America **10**:969, 1930.

50. Tuffier, M.: Hematome sous-peritoneal diffres par rupture spontanei d'un sarcome du rein, Bull. et mém. Soc. d. chirurgiens de Paris **32**:692, 1906.

51. Hartmanns, M.: Spontaneous Rupture of Cancer of the Kidney, Bull. et mém. Soc. d. chirurgiens de Paris **32**:695, 1906.

52. Barney, J. D., and Mixter, W. J.: Renal Tuberculosis Complicated by Inguinal Renal Fistula, Transverse Myelitis and Renal Calculus, J. Urol. **4**:391, 1920.

53. Schwartz, cited by Chazet.[15]

the development of a cutaneous fistula in the loin. Vright [54] discussed a case of advanced bladder and renal tuberculosis complicated by troublesome urinary sinuses which perforated through Petit's triangle. Broca,[55] Lucas [56] and Doyen [57] have mentioned instances of chronic urinary fistula opening in the loin as the result of caseous degeneration of the kidney.

Nephrolithiasis associated with pyonephrosis is often antecedent to perforation of the kidney; the stone is extruded into the perirenal tissue and finally escapes by burrowing its way through the skin. McClelland [58] described a case of ureterolithiasis which caused a renal fistula with two cutaneous openings; the first sinus opened into the loin and continued to discharge pus, urine and stones; four years later a second fistula opened through the groin, giving exit to pus and stones; both fistulous tracts healed following a nephrectomy. Weeks,[59] Gaston [60] and Whately [61] have cited cases of calculous pyonephrosis which terminated by the stones being expelled through the lumbar muscles. Crabtree [62] encountered a perinephritic abscess arising from a calculous perforation of the renal cortex, the escaping urine and pus spreading beneath the diaphragm causing a secondary subdiaphragmatic abscess.

Cutaneous renal fistula occurring as a result of a parasitic infection of the kidney is extremely rare. La Peyre [63] saw an unusual case in which the kidney had been converted into a fatty mass harboring three worms about 3½ inches (8.9 cm.) in length (the type not described). Several worms were found embedded in the perirenal tissue, and a sinus tract leading from the kidney and opening in the loin permitted a continuous discharge of purulent urine.

Cutaneous urinary fistulas also arise as the result of spontaneous rupture of a hydronephrotic kidney. Coupland and Lawton [64] treated a patient with a bifid ureter which was obstructed at the vesical orifice by a large irregular stone; the subsequent urinary stasis caused a pres-

54. Vright, cited by Weiser.[30]

55. Broca, C. D., cited by Chazet.[15]

56. Lucas, cited by Weiser.[30]

57. Doyen, T. B., cited by Chazet.[15]

58. McClelland, cited by Morris.[3]

59. Weeks, S. H., cited by Morris.[3]

60. Gaston, J. M., cited by Morris.[3]

61. Whately, F.: Renal Calculus Which was Discharged Through a Fistulous Opening in the Loin, Tr. Path. Soc. London **26**:128, 1874-1875.

62. Crabtree, E. G.: Calculous Pyonephrosis with Subdiaphragmatic Abscess, Boston M. & S. J. **196**:825, 1927.

63. La Peyre, cited by Morris.[3]

64. Coupland and Lawton, cited by Morris.[3]

sure atrophy with rupture of the renal cortex, and the extravasated urine finally perforated the lumbar muscles. Morris described a similar case, in which the obstruction was due to a ureteral stone.

URETERAL FISTULA

Spontaneous perforation of the ureters is much less common than perforation of the kidney. However, the same pathologic conditions that produce renal fistula are responsible for the great majority of ureteral sinuses.

Uretero-Intestinal Fistula.—The anatomic relationship of the left ureter and the stomach precludes the formation of a gastro-ureteral fistula unless some anomalous condition is present.

Communications between the right ureter and the duodenum have occurred but are rare. Several questionable cases have been reported, but careful study shows that most of them were those of nephro-duodenal fistulas. Davis [65] cited the only authentic case of uretero-duodenal fistula. The patient was 29 years of age, and complained of a dull, aching pain in the left flank associated with fever, chills, pyuria, frequency and burning on urination. A pyelogram revealed the extravasation of the injected medium from the upper end of the ureter into the periureteral tissue. At operation, a sinus tract was found uniting the ureter and the duodenum. The opening in the duodenum was closed, and the kidney was removed. The patient made a good recovery. There was sudden cessation of all urinary symptoms following the operation. The kidney was examined microscopically, and no evidence of tuberculosis could be found. Davis believed that the non-specific perirenal abscess ruptured into the ureter and also emptied into the duodenum, thus forming the artificial communication.

Stones may ulcerate through the ureteral wall and form a peri-ureteral abscess which may empty into the colon. Greene [66] observed a patient in whom dysuria and frequency of urination were outstanding symptoms. Pyelographic studies disclosed a distorted renal pelvis, and at the level of the fourth lumbar vertebra the urographic solution was seen to escape from the ureter and enter the cecum and ascending colon. Operative findings substantiated the diagnosis of ureterocolic fistula. Wells [67] cited the case of a patient who passed urinary calculi in the feces. He concluded that the stones ulcerated through the lower

65. Davis, E. G.: Duodeno-Ureteral Fistula of Spontaneous Origin, J. A. M. A. **70**:376 (Feb. 9) 1918.

66. Greene, L. B.: Uretero-Colonic Fistula, Ann. Surg. **86**:130, 1927.

67. Wells, S.: Urinary Calculus Discharged Through the Rectum, Tr. Path. Soc. London **5**:202, 1854.

ureter, formed a periprostatic and perirectal abscess which ruptured into the rectum, thus permitting urine and stones to escape with the stools.

Retrocecal appendix may cause a perityphlic abscess which may burrow its way into the ureter and empty its purulent contents by means of the urinary tract. LeComte emphasized that such abscesses not only communicate with the ureter, cecum and colon but may open externally by penetrating the lumbar muscles, thus producing a urinary-iliocolostomy. Chrzelitzer [68] also found an appendico-ureteral fistula due to an old appendical abscess.

Most ureterocolic fistulas result from tuberculous infections. Doering [69] treated a patient who had a cold abscess which arose from a tuberculous hip and eroded into the ureter, producing pyuria, and then ruptured the colon. Casper,[70] Newman [71] and Doering each encountered uretero-intestinal fistulas resulting from tuberculous periureteritis. Van Vranken [72] observed a patient with a swelling in the groin just above Poupart's ligament. He incised the abscess and found that it communicated with the colon. Feces drained from the opening for about one month, and then urine could be detected in the discharge. Evidently the psoas abscess had perforated both the colon and the ureter, forming a ureterocolostomy.

Uretero-Abdominal Fistula.—One of the most unusual forms of ureteral fistula is that in which there is a communication with the peritoneal cavity.

Dystrophies of the ureter, whether congenital or acquired, are conducive to rupture of the ureteral lumen. Foster, Pflaum and Sugoi [73] described the case of a child 14 days old who died of urinary ascites. Autopsy showed atresia of the right ureter with an aplastic right kidney. The left ureter had a normal lumen, but just proximal to its vesical orifice the ureter was kinked, producing complete obstruction. The ureter was dilated and tortuous, and was the seat of an advanced ureteritis. Just above the obstruction was a small perforation of the ureteral wall which permitted the escape of urine into the peritoneal cavity.

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68. Chrzelitzer: Beitrag zur Hämaturie nach Appendizitis, Ztschr. f. Urol. **16**:408, 1922.

69. Doering, cited by Weiser.[30]

70. Casper, L.: Ureteral Fistula and Ureteral Suppurations, Ztschr. f. Urol. **18**:545, 1924.

71. Newman, cited by Weiser.[30]

72. Van Vranken, A. T.: Uretero-Intestinal Fistula as Result of Abdominal Abscess not Due to Appenditicis, with Recovery, Tr. New York M. A. **11**:553, 1894.

73. Foster, Pflaum and Sugoi, cited by Weiser.[30]

Tuberculous ureteritis with its inflammatory necrosis may perforate the ureter and empty its purulent contents into the abdominal cavity, producing the clinical picture of peritonitis. Mitterstiller [74] performed a laparotomy for chronic peritonitis, on a woman, 28 years of age, and found multiple intraperitoneal and extraperitoneal abscesses caused by the tuberculous ulceration of the ureter at the level where it crossed the iliac vessels. The findings were corroborated at necropsy.

In case 5 of our series the ureterovesicoperitoneal fistula was due to an extensive tuberculous necrosis of the bladder and ureter. The urine secreted by the left kidney entered the peritoneal cavity either by the ureteroperitoneal or by the vesicoperitoneal fistula, and then escaped from the abdominal cavity by means of an old lumbar sinus.

A calculus may partially or completely obstruct the ureter, causing not only a hydro-ureter but also ureteritis, both of which weaken the walls of the ureter and may cause a perforation. Dupuytren [75] observed the case of a young woman with a left lumbar abscess, which was incised and drained. Subsequently, signs of chronic peritonitis developed, and the patient died. At autopsy, it was found that a stone had eroded through the musculature of the ureter and permitted the infected urine to seep out into the peritoneal cavity.

As these histories indicate, most cases of urinary ascites terminate fatally owing to peritonitis precipitated by the contaminated urine.

Ureteroperiureteral Fistula.—Stones may ulcerate their way through the walls of the ureter producing a periureteral abscess which drains into the ureteral lumen, causing pyuria. Frenkel [76] gathered the records of eleven cases of this type. Berry [77] encountered a phlegmonous involvement of the periureteral tissue due to urinary extravasation and found that a small stone had perforated the ureter. Schmidt [78] treated a patient with periureteritis which was secondary to perforation of the ureteral wall by a stone. The patient had symptoms suggesting carcinoma of the cervix, and only after careful study was it found that the extensive periureteritis was due to urinary extravasation.

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74. Mitterstiller, S.: Ueber einem Fall von Entleerung einer Pyonephrose in de Peritonealhohle, Ztschr. f. Urol. **14**:168, 1920.

75. Dupuytren, cited by Morris.[3]

76. Frenkel, cited by Weiser.[30]

77. Berry, J.: Perforation of Ureter by Calculus: Extravasation of Urine, Brit. J. Surg. **8**:372, 1921.

78. Schmidt, H. H.: Perforation of Ureter by Calculous Invisible Roentgenographically and Causing Symptoms Resembling Cancer Uterus, Zentralbl. f. Gynäk. **54**:274, 1930.

Diverticula of the ureter, which usually represent congenital dystrophy, may rupture spontaneously. Stevens [79] observed the case of a man, 59 years of age, who complained of fever and chills and pain in the left lower flank; urinary symptoms were entirely absent.

Cystoscopic and pyelographic examination revealed a small diverticulum at the ureteropelvic junction; the diverticulum had perforated and was draining a coexisting perinephritic abscess.

Perinephritic abscesses resulting from osteomyelitis or infections of the adjacent soft tissues may rupture into the ureter and cause a fistula. Lindsjö [80] cited the case of a patient who suffered for eighteen months from intermittent pyuria and pains in the left hip. A specimen of urine collected from the left ureter was loaded with pus. At operation a large psoas abscess was found which had arisen from a chronic osteomyelitis of the hip bone. The abscess had penetrated the walls of the ureter and was emptying its purulent contents into the urinary stream.

Tuberculous ureteritis with perforation and formation of periureteral abscesses may closely simulate some intra-abdominal lesions. Rathbun [81] encountered a scrofulous periureteral abscess which communicated with the left ureter in a patient in whom the referring physician had suspected an extra-uterine pregnancy.

Our case (case 4) of ureteroperiureteral fistula illustrates the complications that may follow a perinephritic abscess. The kidney was the seat of cortical abscesses; the infection spread to the perinephrium, extending down the course of the ureter and forming a periureteral abscess which ruptured into the ureter, causing cystitis with pyuria.

Ureterovesical Fistula.—Abnormal spontaneous communications between the ureter and bladder are unusual. Occasionally a ureteral stone may be arrested at the vesical orifice, ulcerate its way through the walls of the ureter and form a periureteral abscess which may penetrate the bladder. Occasionally a pelvic abscess secondary to acute salpingitis ruptures into both the ureter and the bladder, forming a ureterovesical fistula, such as Ottow [82] described. Schottelius [83] treated two patients suffering from periureteritis. The inflammation had spread

79. Stevens, A. R.: Diverticulum of Ureter: Acute Inflammation and Spontaneous Perforation, J. Urol. **16**:157, 1926.

80. Lindsjö: Case of Pyuria with Unusual Etiology, Acta pædiat. **4**:104, 1925.

81. Rathbun, N. P.: Necrosis of the Ureter, Perforation, Peri-Ureteral Abscess, J. Urol. **17**:329, 1927.

82. Ottow, B.: Rupture of Abscess of Internal Genitalia into Bladder and Ureter, Zentralbl. f. Gynäk. **53**:2551, 1929.

83. Schottelius, M.: Zwei Fälle von einseitiger Hydronephrose (Ureterovesicalfistel), Virchows Arch. f. path. Anat. **71**:268, 1877.

to the ureters, producing ureteritis and hydro-ureter. The diseased ureteral walls ruptured, permitting urine to escape into the periureteral tissue. The resulting abscess opened into the bladder and formed a uretrovesical fistula.

Ureterovaginal Fistula.—Fistulous tracts joining the vagina and the ureters as the result of local disease are very interesting. Weinzierl [84] referred to a case in which a fistula arose from the prolonged use of a pessary. The constant irritation of the pessary with its inflammatory reaction and pressure necrosis produced localized abscesses which opened both into the vagina and into the ureter, permitting urine to escape through the vulva. Morris treated a young girl for renal tuberculosis and cystitis. The bladder symptoms became so pronounced that the patient dribbled urine constantly. On careful examination, an advanced tuberculous ureteritis with a cold abscess was found. The inflammatory process had extended to the periureteral tissues and finally had eroded its way into the vagina. All the urine excreted by the isolateral kidney escaped by this fistulous tract. Jurgen [85] and Kretchmer [86] each described a case of ureterovaginal fistula arising from irradiation necrosis following the application of radium for carcinoma of the cervix.

An interesting case was described by Konwer [87] and Schwartz [83] in which a stone had ulcerated its way through the coats of the ureter and had formed a periureteral abscess which had perforated the walls of the vagina.

DIAGNOSIS

In the majority of the cases cited, the diagnosis was made from the history and the findings at operation or autopsy. At the present time, cystoscopic and roentgenographic studies make the diagnosis of urinary fistula relatively easy, and there is no reason why a preoperative diagnosis of all such fistulas should not be made.

TREATMENT

The best treatment for spontaneous urinary fistula is prophylaxis. The infrequent occurrence of this condition at the present time signifies that, in general, renal diseases are not allowed to progress to the stage at which fistulas are formed. Tuberculous kidneys usually are removed before they reach the point of pronounced caseous degeneration; stones are extracted before they penetrate the walls of the pelvis or ureter;

84. Weinzierl, cited by Weiser.[30]
85. Jurgen, O., cited by Weiser.[30]
86. Kretchmer, cited by Weiser.[30]
87. Konwer, cited by Weiser.[30]

perinephritic abscesses are drained before they ulcerate into the kidney, and hydronephrotic kidneys are prevented from rupturing by early removal of the ureteral obstruction and the establishment of free drainage. If urinary fistulas develop, it usually means that the patient has refused medical attention until the disease process is well established.

The therapeutic measures employed in the treatment of spontaneous renal fistulas are governed by the general condition of the patient, the functional status of the kidneys, the type of disease present and the organs or tissues involved. Attempts to secure a primary closure of the sinus tract usually are unsuccessful unless the causative factors are removed. As such cases represent advanced renal tuberculosis, nephrolithiasis, pyonephrosis or hydronephrosis with atrophy of the parenchymal tissue, it is generally necessary to sacrifice the kidney.

Many patients of this type represent such bad surgical risks that a primary nephrectomy is contraindicated; however, an incision and drainage of the coexisting perirenal abscess usually improves the general condition so that the kidney may be removed at a later date with much less danger. In our case (case 2) of nephrocolic fistula such a conservative procedure was followed, and in spite of three operations the patient made a satisfactory recovery. At times the chronic infection associated with most renal fistulas has so reduced the vitality of the patient that even the most conservative surgical procedure is of no avail.

If the fistula communicates with a hollow viscus, such as the gastrointestinal tract, the opening in the cavity is closed and the kidney is removed. Very few fistulas persist after the kidney has been excised, unless the initial disease has become firmly established in the perirenal tissues.

The treatment of ureteral fistulas is usually more conservative, depending entirely on the condition of the isolateral kidney. If a small stone has perforated the ureter, incision and drainage of the periureteral abscess and the use of indwelling ureteral catheters sometimes result in cure. If pyonephrosis has developed secondary to ureteral obstruction, the kidney usually must be sacrificed. In our case of ureteral-periureteral fistula, which was secondary to pyonephrosis associated with a perirenal abscess which had ruptured into the ureter, the removal of the kidney was necessary in order to close the fistula. In most patients with renal or ureteral fistulas, the condition constitutes a complication arising from advanced kidney disease and is attended with a high rate of mortality. The infected urine infiltrates all the surrounding tissues and organs, producing chemical and bacterial irritations which are expressed by severe febrile and septic reactions. In our series of four patients who were subjected to operation, one death occurred.

SUMMARY

This report includes the detailed study of three types of spontaneous renal fistulas; nephrocolic, nephroperirenal and renochylous fistula, and one case each of spontaneous ureterovesicoperitoneal and ureteroperiureteral fistula.

Spontaneous renal and ureteral fistulas are the result of advanced kidney disease, such as tuberculosis, nephrolithiasis, pyonephrosis, hydronephrosis or neoplastic disease, and occasionally arise from a perinephritic abscess that is secondary to caries of the vertebrae, perityphilic abscess, pelvic disease or peripheral infections, such as boils.

The diagnosis is made on the basis of the clinical symptoms, corroborated by cystoscopic and roentgenographic studies.

The most important consideration in the treatment of spontaneous urinary fistulas is prophylaxis, the prevention of advanced kidney diseases to the stage of fistula formation by the use of early corrective measures. Closure of a renal fistula usually requires nephrectomy, as the kidney is so generally diseased that practically no functioning tissue remains. In the case of patients who constitute a grave surgical risk, conservative measures are indicated; primary drainage of a perinephritic abscess usually results in improvement, so that a nephrectomy may be performed later. The use of indwelling ureteral catheters may facilitate the repair of a ureteral fistula, but usually the kidney is harboring infection which has destroyed the parenchyma to such an extent that its conservation is useless.

EFFECT OF COMPLETE AND PARTIAL STARVATION ON THE RATE OF FIBROPLASIA IN THE HEALING WOUND

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The effect of complete and partial starvation on the healing of wounds is of fundamental importance to the surgeon. Malnourished patients must occasionally be subjected to surgical procedures, and often it is necessary to restrict the amount of diet after operation. Only too frequently we observe the wounds of undernourished patients, particularly those of children, healing slowly and becoming infected.[1] Unfortunately, in the clinic, however, it is not always possible to establish the exact nature of a malnutrition or to determine the deficiency of the previous diet which produced it. So, too, it is quite impossible to tell whether a prolonged healing time of a wound is the result of malnutrition alone or of other causes more obscure. To interpret adequately the relation of diet to the healing wound, one must have carefully collected data on experimental animals—data which give the preoperative state of nutrition and the dietary regimen as well as the postoperative state.

Repeated experiments have shown that starvation actually accelerates the rate of cellular division. Morgan,[2] for example, found that the legs of salamanders regenerated as rapidly when the animals were starved as when they were well nourished. Gaglio,[3] studying the problem histologically, found mitotic figures more frequent in the

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1. Harvey, Samuel C.: Post-Operative Complications, in Nelson Loose-Leaf Living Surgery, New York, T. Nelson & Sons, 1927, vol. 1, p. 552.

2. Morgan, Thomas H.: The Physiology of Regeneration, J. Exper. Zool **3**:457, 1906.

3. Gaglio, G.: Sulla alterazioni istologiche e funzionali dei muscoli durante l'inanizione, Arch. di sc. med. **7**:301, 1884; cited by Morgulis.

tissues of starved frogs than in normal frogs, while Morpurgo [4] saw a similar increase in cells of pigeons. Morgulis,[5] following a careful study of the entire problem, came to believe that a reduction in cell cytoplasm occurred, with but little reduction in the size of the nucleus. This disturbed nuclear cytoplasmic ratio, he believed, resulted in an increased rate of cell division in an effort to restore the proper balance. Theoretically, then, starvation should not delay healing of the wound, but actually, as has been mentioned, there are disturbing factors in association with complete or partial starvation which cause the wounds to show not an acceleration in healing, but rather a slower rate of repair. Consequently, it is not surprising that it is a common belief that complete or partial starvation delays the healing of the wound, and this may be true because regeneration is only one phase of the healing.

It is the object of our investigation, therefore, to dissociate starvation and partial starvation (reduction in the amount of an adequate diet) from other types of malnutrition and to study their effects on the healing wound. To a certain extent, the strength of the wound has been found to be directly proportional to the degree of healing attained, and, therefore, strength will be adopted here as the criterion for testing the rate of repair. In order to determine the relation between age and these two dietary changes, it will be necessary to make two studies, using, on the one hand, adult animals and, on the other, young ones.

METHOD

The rat was particularly good for our purposes because of its relatively high degree of immunity to infection, as well as to the large amount of work that has been done on its metabolism.

Because we found that age made such a difference in the rate of healing,[6] young and old rats of definite specifications were selected. The adults employed averaged 194 Gm. in weight and were at least 5 months old. They were kept in individual cages. The quantity of the adequate diet given to the controls was determined by the amount of the same diet consumed by a normal group of rats in a previous experiment (tables 1 and 2). This was on the average 3.4 per cent of the body weight daily. The partially starved rats were given one-half this amount, or 1.7 per cent daily, and those completely starved were given only water. In order

4. Morpurgo, B.: Sull processo fisiologico di neoformazione cellulare durante la inanizione acuto dell' organismo, Arch. di sc. med. **12**:395, 1888; cited by Morgulis.

5. (*a*) Morgulis, S.; Howes, P. E., and Hawk, P. B.: Studies on Tissues of Fasting Animals, Biol. Bull. **28**:397, 1915. (*b*) Morgulis, S.: Fasting and Undernutrition: A Biological and Sociological Study of Inanition, New York, E. P. Dutton & Co., 1923.

6. Howes, E. L., and Harvey, S. C.: The Age Factor in the Velocity of the Growth of Fibroblasts in the Healing Wound, J. Exper. Med. **55**:577 (April 1) 1932.

to establish definitely the malnutrition before the wound was inflicted, the partially starved animals were placed on the diet one week before operation, but those completely starved were for obvious reasons not placed on their water diet until the day before operation.

The young rats selected were under 2 months of age and weighed 78 Gm. on the average. The controls of this group were given a normal dietary intake of 13 per cent of the body weight daily (table 2), and consequently those partially starved received 6.5 per cent daily. Again, those completely starved were given only water. It will be noted that these young rats were given four times the quantity of the ration allowed the adults, but both groups received equivalent amounts in proportion to their requirements. Young rats need four times as much

TABLE 1.—*Standard Balanced Diet (Smith and Moïse)*

Percentage Composition		Calories per Kg. of Food	Apportionment of Total Calories, per Cent	
Casein	18	738	Protein	13.8
Starch	51	2,091	Carbohydrate	39.2
Crisco	22	2,046	Fat	47.0
Cod liver oil	5	465		
Salts (Osborne and Mendel mixture)	4			
		5,340		
Lettuce twice a week 70 mg. of yeast daily				

TABLE 2.—*Consumption of Food and Variation in Weight of Young and Adult Rats on the Standard Diet* *

	Adult Rats		Young Rats	
	Start to Operation	Operation to Death	Start to Operation	Operation to Death
Difference in weight per day, per cent	+0.26	−0.45	+4.1	+3.1
Food consumed per day, per cent	4.3	2.5	12.0	11.0
	Av. 3.4		Av. 11.5	
Protein consumed per day, per cent	0.75	0.45	2.2	2.0
Calories of food consumed per day, per cent	23.0	13.0	59.0	55.0
Calories of protein consumed per day, per cent	3.1	1.7	8.1	7.5

* The calculations are based on the percentage of body weight at the start of the experiment. The data are compiled after Towes and Harvey.[6]

food as adults (table 2). The weights of both the adult and the young rats completely starved were recorded daily, while those only partially starved were weighed every third day.

The details of wounding the stomachs and of testing the healing strengths at regular intervals have been given elsewhere.[7] Essentially, it consisted in making a wound 1 cm. in length through the wall of the cardiac or rumen portion of the stomach. This was subsequently sutured in two layers with no. 000 plain catgut, approximating first the squamous cell layer and then the muscularis and serosa. After the animal was killed at the given postoperative interval, the strength of the wound was determined by finding the amount of air pressure necessary to rupture it. The machine developed for these observations was the same as that used in former studies.[8]

7. Harvey, S. C., and Howes, E. L.: Effect of High Protein Diet on the Velocity of Growth of Fibroblasts in the Healing Wound, Ann. Surg. **91**:641 (May) 1930.

8. (*a*) Harvey, S. C.: The Velocity of the Growth of Fibroblasts in the Healing Wound, Arch. Surg. **18**:1227 (April) 1929. (*b*) Harvey and Howes.[7]

Postmortem studies were made not only of the wound but of the organs of the body. Microscopic sections were prepared where deemed necessary.

In the previous investigations it was found that the curve of strength of the wounds plotted at daily intervals showed three distinct phases of healing (chart 1). The first phase was marked by a latent period of four days during which the wound had only the strength imparted to it by the sutures and the deposited fibrin. During the second phase, from the fourth to the tenth day, fibroplasia developed and strength increased rapidly. In the third or final phase, from the tenth to the fourteenth days, some of the wounds became stronger than the surrounding tissues, and consequently, the points of rupture were elsewhere than in the wound. On the final day of this third phase, all of the wounds became stronger, and, therefore, this day was considered the end-point. Because we found that the presence of the sutures caused variability in the strength determinations during the first phase, in this experiment we did not seek the readings over this interval. Likewise, we omitted

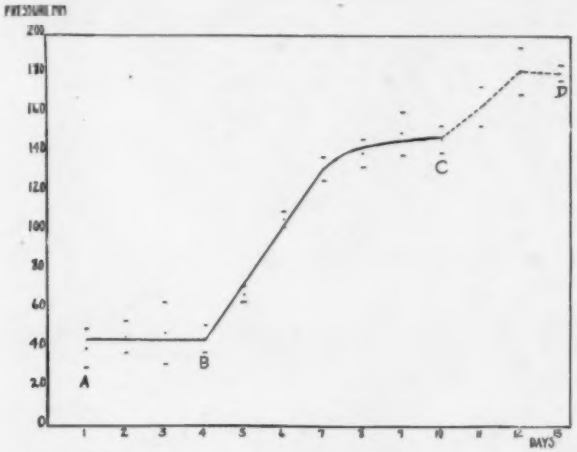

Chart 1.—Typical healing curve for wound of the stomach: A to B, period of fibrinous healing; B to C, period of fibrous healing, ruptures in the wounds; C to D, continuation of fibrous healing, ruptures in the wounds and elsewhere in the wall of the stomach.

the final period for many readings were obtained in this phase which gave the strength of the wall of the stomach and not that of the wound. It was deemed highly important, however, to note the day on which rupture of the stomach other than at the site of the wound began to occur. As a result, our determinations concern themselves only with the phase of fibroplasia. In order to reduce individual biologic variations undoubtedly existent, the strengths of five different wounds in the stomach were averaged for each twenty-four hour postoperative interval from the fifth to the tenth days, inclusive. With these averages, curves were constructed in the manner previously described.[8a]

RESULTS AND COMPARISON OF THE CURVES

Adult Rats.—A. Completely Starved Group (table 3): The adult rats withstood complete starvation until the tenth day; one died on the eighth day, two on the ninth and four on the tenth. The one remaining alive on the tenth day was extremely weak and lethargic.

The curve for healing strength of their wounds was somewhat different than those for the other adult rats (chart 2). All the curves were practically the same until the fifth day, but on the following two days the rate of ascent of the curve for starvation fell below the other two curves. This decrease in the rate was of short duration, however, for on the eighth and ninth days of healing, the curve again mounted more rapidly and the strengths registered were greater than those of the rats receiving the half caloric diet and the full diet. During the entire nine days of starvation, the final phase of the curve for healing strength was not reached, but then this period was not gained in the

TABLE 3.—*Average Strengths of Stomach Wounds for Adult Rats**

Post-operative Days	On Standard Diet		With Starvation		With Partial Starvation	
	Mm. of Mercury	Standard Deviation of the Mean	Mm. of Mercury	Standard Deviation of the Mean	Mm. of Mercury	Standard Deviation of the Mean
5	71.0	± 5.0	72.0	± 6.0	69.0	± 7.0
6	107.0	± 6.0	93.0	± 6.0	96.0	± 6.4
7	135.0	± 5.0	133.0	±11.0	106.0	± 6.0
8	143.0	± 6.8	161.0	±10.0	147.0	± 9.0
9	153.0	±10.0	171.0	± 8.0	154.0	± 6.0
10	150.5	± 7.0	Death		155.0	±10.0
	Final period				Final period	

* All of the breaks occurred in the wounds.

TABLE 4.—*Percentage Loss in Body Weight of the Adult Starved Group*

Postoperative Days	Percentage	Postoperative Days	Percentage
1	7.8	6	6.9
2	6.2	7	4.5
3	5.6	8	4.0
4	5.7	9	3.0
5	5.7	10	2.8

curve representative of the full diet until the tenth day. It can be concluded, therefore, that although this curve for starvation was somewhat different in form than the curve for the controls, yet in general the rate of healing of these wounds was not markedly changed from the normal rate.

Determinations of the losses in weight of these rats calculated from the weight at the onset of the study showed that the average percentage loss of body weight was more rapid during the first day (7 per cent), and that the amount lost decreased daily as starvation progressed (table 4). These rats drank only a minimum amount of water. At autopsy, the incidence of wound infection in this group was but 2 per cent, or identical with that found in the controls. The postmortem observations substantiated the observations on loss of weight, for in every case body fat was either entirely absent from the usual depots

or so reduced in amounts as to be found with difficulty. All skeletal muscles were in an advanced state of atrophy, and practically every stomach contained a large hair ball.

B. Partially Starved Group: The curve of healing in these animals did not differ markedly from the curve for the controls, except for some slowing of the rate on the sixth and seventh day (table 3 and chart 2). The average daily loss in body weight was appreciably less than in the completely starved group, being here but 3 per cent or about

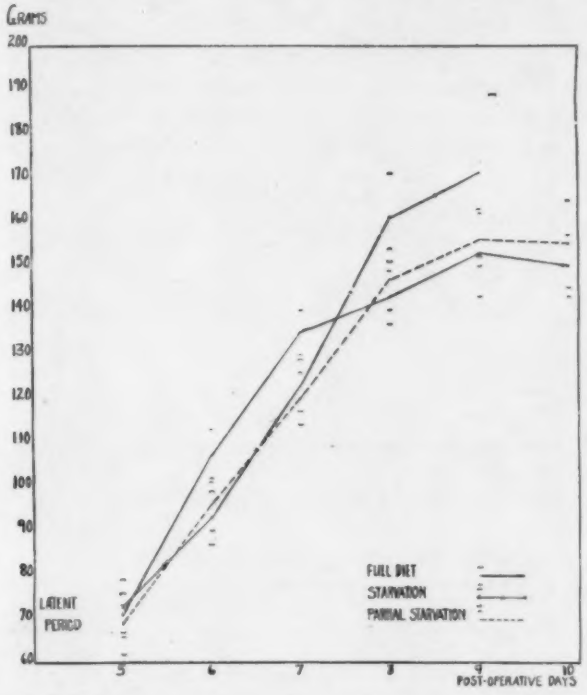

Chart 2.—Strength of stomach wounds for adult rats. Note the greater rate of healing on the seventh to the eighth days in the curve for starvation.

half that observed on complete starvation. The incidence of infection was, however, higher than that found with starvation, 8 per cent being found in the abdominal wounds and 3 per cent in the peritoneal cavities. At autopsy, more fat was observed in the depots, and there was decidedly less muscular atrophy than had been found in the completely starved animals. Taking the progress of healing in its entirety, partial starvation did not retard the rate of the healing of the wounds in these adult rats.

Young Rats.—A. Completely Starved Group (table 5): The young rats did not withstand complete starvation long enough for their wounds

to give satisfactory determinations of healing strength. None of the animals lived longer than the fifth day postoperatively, and the majority died on the fourth day. Wounds tested in two surviving animals on

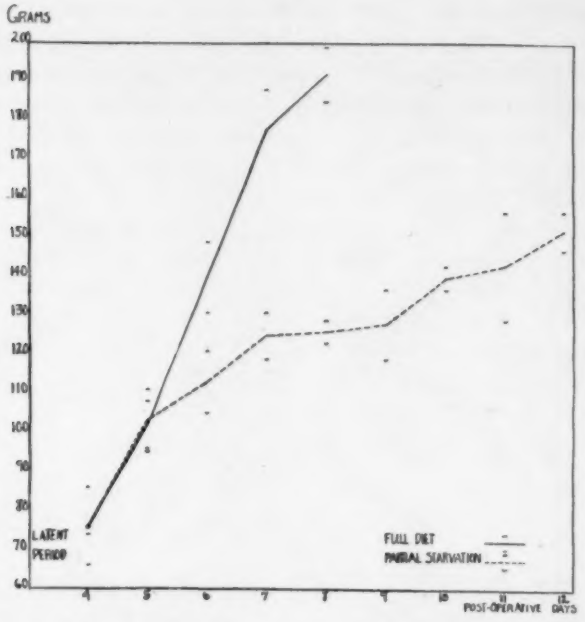

Chart 3.—Strength of stomach wounds for young rats.

TABLE 5.—*Average Strengths of Stomach Wounds for Young Rats**

Post-operative Days	On Standard Diet		With Starvation		With Partial Starvation	
	Mm. of Mercury	Standard Deviation of the Mean	Mm. of Mercury	Standard Deviation of the Mean	Mm. of Mercury	Standard Deviation of the Mean
4	76.0	± 2.0	78.0	± 3.0	76.6	± 9.5
5	102.0	± 6.0	Death		103.7	± 8.0
6	140.0	± 9.0	.....		113.2	± 8.0
7	178.0	±10.0	.....		125.6	± 6.7
8	192.0	± 7.0	.....		126.0	± 3.7
9	Final period		.....		128.3	± 9.0
10	.....		.....		140.5	± 3.7
11	.....		.....		143.2	±14.0
12	.....		.....		152.0	± 5.7

* All of the breaks occurred in the wounds.

the fourth day had only the strength given to them by the sutures and fibrinous deposits. There was no evidence of fibroplasia microscopically.

B. Partially Starved Group: The plotted determinations of the healing strength of the wounds for this group had the same general trend as the controls until the fifth day (table 5, chart 3). After this

day, however, the rate of repair fell off rapidly, and the increase in strength of the wounds from day to day was so small that the curve tended to flatten out. Healing was so retarded that the entrance of the curve into its final period was not reached during thirteen days of testing. In the last few days the strength even fell below that for the adult control. This was an astounding result for the rate of fibroplasia in young rats has always been found to be greater than in adult rats.[8]

Unlike the adults who had shown but little desire to move about, these young animals were very active. In fact, at times it seemed as though they were in a state of tetany. The slightest noise would cause them to jump to the tops or sides of their cages. They also drank more water than the controls, and despite an average daily loss in body weight of 1.9 per cent, they continued to grow in body length (table 6). The incidence of wound infection was 5 per cent both in the abdominal wall and in the peritoneal cavity.

TABLE 6.—*Weight Data for Young Rats Partially Starved*

	Initial Body Weight, Gm.	Final, Gm.	Per Cent Lost Daily	Daily per Cent Weight Lost
Four days	76	71	6.6	1.6
Five days	76	66	13.2	2.6
Six days	71	60	15.5	2.6
Seven days	77	65	15.6	2.2
Eight days	55	45	18.2	2.2
Nine days	86	71	17.4	1.9
Ten days	71	57	19.7	1.9
Eleven days	86	66	23.3	2.1
Twelve days	78	71	9.0	0.8
Average daily per cent weight lost				1.9

COMMENT

The comparison of the curves for the wounds of the adult rats indicates that the rates of fibroplasia were practically the same under all three dietary conditions. This proximity of the three curves demonstrates that an acute state of malnutrition can exist in adult rats without appreciably affecting the rate of repair of soft tissue. It is highly probable that the explanation of this ability of the wound to heal in spite of caloric deficiencies of the diet lies in the compensating mechanisms of metabolism known to occur in association with inanition.[9] The time required for exhaustion of the various essential food elements during complete or partial starvation depends on (1) the amounts of these substances consumed in the diet, (2) the amounts available within the body as stored reserves and (3) the rapidity with which they

9. Benedict, F. G.: A Study of Prolonged Fasting, Washington, D. C., Carnegie Institute, bull. no. 203, 1915. Jackson, C. M.: The Effects of Inanition and Malnutrition upon Growth and Structure, Philadelphia, P. Blakiston's Son & Co., 1925.

are consumed. The exact character of the effect of complete or partial inanition, then, is exceedingly variable in a group of animals. Exhaustion of all essential food elements at the same time is absolutely impossible. It is recognized that for a varying period of time the nutrient level of the blood is maintained by the mobilization of foodstuffs from the reserve depots. The glycogen of the liver is said to be utilized first; secondly, the depot fat, and lastly, the proteins in the tissues. The extent of metabolic adjustment is dependent in part on the caloric insufficiency of the diet; consequently less metabolic adjustment was necessary in the partially starved rats and there was found less depletion of body fat and no atrophy of the muscles. This was borne out by the losses in weight, for the greater losses were in the completely starved rats.

The irregularity of the rate of ascent of the curve for starvation in the adult rats suggests that a multiplicity of factors affected it. The selection of the animals with their variations in their original food reserves might offer a possible explanation for this irregularity. But even if this selection were given all the credit due to it for maintaining the rate of repair, the important conclusion cannot be escaped that in general the rate of healing in adults was not decreased by starvation. This point is particularly well emphasized by the degree of healing strength attained by the wounds of the animals in extreme starvation on the eight and ninth day.

Studies on protein metabolism during starvation have shown that in the first two days of complete fasting the excretory products of nitrogen increase, but subsequently they establish themselves at a constant level. In the final days of starvation, the metabolism is almost all if not entirely protein, for there is again a definite increase in the rate of tissue destruction, and during this period, therefore, there would be an increase in the available amounts of amino-acids. It is more than pure theory, then, when we assume that the increased rate of healing during the final days of starvation was caused by this accelerated metabolism of proteins. To substantiate this there is the work of Carrel and Baker,[10] who have shown that in tissue cultures proteins are the necessary stimulants for cell proliferation, and Clark [11] and Harvey and Howes [7] have demonstrated that an increased quantity of proteins in the diet stimulates the rate of healing of the wound.

Another possible explanation for the terminal increase in the rate of healing of the wounds in the completely starved adult rats may lie in the changes in the acid-base equilibrium known to occur during the

10. Carrel, A., and Baker, L. E.: The Chemical Nature of Substances Required for Cell Multiplication, J. Exper. Med. **44**:503, 1926.

11. Clark, A. H.: The Effect of Diet on the Healing of Wounds, Bull. Johns Hopkins Hosp. **30**:117, 1919.

concluding days of starvation. Although true acidosis has never been satisfactorily demonstrated in rats, the starvation they underwent undoubtedly reduced their alkaline reserve, and if such is the case, it would seem that the findings of Herrmannsdorfer [12] would explain the increased rate of repair. Herrmannsdorfer found that if an alkaline diet was given to patients with wounds, healing was delayed, while the reverse was true with an acid diet and when there was partial acidosis.

In addition, the disturbed nuclear ratio must not be forgotten as a possible factor stimulating healing of the wound during these terminal days of starvation. We have mentioned before the theory that starvation increases the rate of cellular division by decreasing the nuclear-cytoplasmic ratio. Cells in which this change has taken place approach an embryonic state wherein the cytoplasm is quantitatively reduced while the nuclei remain unchanged. The result is an accentuation of mitosis in an effort to restore the ratio.

Exactly which of these factors controlled the increased rate of healing in the last two days of starvation, we do not know. It is evident, however, that the increased rate during the final two days was caused by some disturbed mechanism produced by complete starvation, for it did not occur with partial starvation. Perhaps, though, we are giving too much credit to the rôle of the general metabolism in the healing of wounds. We have not forgotten the importance of local conditions in the healing wounds, but wish only to emphasize the influence of the general metabolism. This point of view is supported by the works of Clark [11] and Herrmannsdorfer.[12]

Some may consider that the stomachs of the completely starved rats were more at rest than those of the partially starved rats, and that thus an inhibiting factor was removed allowing cells of less proliferative power to heal at a normal rate. That cells do not have less but actually more proliferative power during starvation we have already mentioned. Besides, Howes [13] has shown that it is not so much freedom from function that aids healing as freedom from irritation. In the stomachs of these completely starved rats we found hair balls, and it has already been postulated by Pappenheimer and co-workers [14] that

12. Herrmannsdorfer, A.: Ueber den Einfluss der Nahrung auf die Pufferkapazität des Blutes und den Heilverlauf und Keimgehalt granulierender Wunden, Deutsche Ztschr. f. Chir. **200**:534, 1927.

13. Howes, E. L.: Der Einfluss der Nahrung auf den Heilungsablauf von Magenwunden, Beitr. z. path. Anat. u. z. allg. Path. **88**:435, 1932.

14. Pappenheimer, Alwin M., and Larimore, Louise D.: The Occurrence of Gastric Lesions in Rats: Their Relation to Dietary Deficiency and Hair Ingestion, J. Exper. Med. **40**:719 (Dec. 1) 1924.

hair in the stomach produces sufficient irritation to cause ulcerative lesions, providing there is some deficiency of the diet. Yet the presence of these hair balls with their possible irritation and distention of the stomachs did not retard healing. The difference in the amount of rest, then, did not allow cells of less proliferative power to heal at a normal rate. It is true that rest is of great importance in wound healing in general, but one which we are inclined to discount to a large extent as an influencing factor in this study.

The fact that the different diets did not influence the rate of healing emphasizes the great range in the amount of food in the stomach over which wounds in this organ will heal. While on the one extreme healing was not appreciably changed by starvation, yet on the other it was not impeded by the digestion of a full diet. Clinically, this has been known for some time. In America it is the practice in gastric cases to reduce the diet markedly postoperatively over a longer period of time than in most European clinics. In France and Germany the ration is more liberal, and the patients are returned to their normal diet much more quickly than they are in this country. Both methods can be used because of this ability of the wounds of the stomach to heal under great variations in the amount of food contained. Of course, the postoperative dietary regimen would be most ideal if it would maintain the nutritional state of the patient at an optimum. However, the one drawback to a rapid return to a normal diet is the presence of infection. McWhorter, Stout and Lieb[15] found that infected wounds of the stomach and intestines leaked along the suture lines with the slightest increase of intravesicular pressure. Even the breaking strength of an intact diseased bowel, Senn found,[16] was reduced to 1½ pounds (0.75 Kg.) from the normal of from 6 to 10 pounds (2.7 to 4.5 Kg.) per square inch established by Andrews.[17] Early feedings, therefore, should be no larger than absolutely necessary, if infection is suspected.

Attention must be called to the fact that the malnutritions established in these experiments were caused by complete starvation and by partial limitation of an adequate diet over a short period of time. The results, therefore, are only applicable to these two conditions, not to malnutritions arising from other causes. Our conception of the optimum diet for growth is rapidly evolving, and perhaps this diet which we thought adequate here may later prove to be entirely inade-

15. McWhorter, J. E.; Stout, A. P., and Lieb, C. C.: The Process of Repair in Wounds of the Small Intestine, Surg., Gynec. & Obst. **23**:80 (July) 1916.

16. Senn, Nicholas: Experimental Surgery, Chicago, W. T. Keener, 1889.

17. Andrews, E. Wyllys: Pneumatic Rupture of the Intestine, A New Type of Industrial Accident, Surg., Gynec. & Obst. **12**:63, 1911.

quate. Moreover, we are only beginning to know what constitutes the optimum diet for the healing of wounds.

The effect of complete and partial starvation on the healing of wounds in young rats was quite different from that found in the adults. With complete starvation, life was not maintained long enough for satisfactory determination of the breaking strength of the healing wounds. With partial starvation, healing progressed at the usual rate until the fifth day, and then it definitely became slower. As a result, the entire length of healing time was prolonged.

The explanation of this slowing of the healing rate after the fifth day is difficult. In the adult animals the deficiency of the diet given was compensated for by changes in the endogenous metabolism. Of course, there must have been similar compensating mechanisms in the young. However, because of the greater demands for food for growth, the available food depots were more quickly depleted. Yet, even with this greater demand it is difficult to imagine that the slowing of the rate of healing was caused by a general change in the compensating mechanisms diverting the food substances away from the wound and toward the general maintenance of the body. In the adult rats, it will be remembered there was no such change and healing progressed at the usual rate even until the entire endogenous metabolism was exhausted. A more fitting explanation, then, would be an exhaustion of some food elements or element capable of stimulating and maintaining cellular proliferation, yet not effective during starvation. The answer to these criteria would be a deficiency of vitamins. A deficiency of vitamin B could have been easily established within the time limits of these experiments; it would not have existed during complete starvation, and would not have appeared in such a short time in the adult rats during partial starvation. Besides, the state simulating tetany described for these animals when partially starved is quite similar to the state described for early vitamin B deficiency. Roegholt [18] was disappointed in his efforts to produce change in the rate of healing of epithelial wounds during vitamin A deficiency, but even if he had found such a change, a deficiency of vitamin A could not have been established in less than thirty or forty days. A vitamin A deficiency, then, can be fairly well ruled out. Of course, the relation of the partial deprivation of other food substances in the diet, the proteins and salts, is difficult to evaluate, particularly in their relation to vitamin deficiencies. As far as we know a partial deficiency of proteins or salts does not bring about a difference in the rate of healing because of the compensating

18. Roegholt, N.: Effect of Vitamin A on Myelopoiesis and the White Blood Picture and Its effect on Wound Healing, Nederl. tijdschr. v. geneesk. **2**:3744 (Aug. 10) 1929.

endogenous metabolism, but because the proteins are so necessary for growth and for healing of the wound, and because they are not utilized during a vitamin deficiency, an interrelation of these two food substances is suggested.

We give no apology for this theorizing over the relation of healing of the wound and vitamin deficiencies, because we are, at present, investigating the effect of the vitamins on the basis of this theory. Later we hope to have some definite proof.

Because of the recognized difficulty of interpreting the significance of infections of the wound in rats, a consideration of the different incidences observed on the three diets studied has purposely been omitted. The data were simply accumulated as a matter of routine.

SUMMARY

In adult rats the rate of return of healing strength in wounds of the stomach was not appreciably affected by complete starvation. Neither was it affected by giving one-half the required amount of an adequate diet over a short period of time. On the other hand, the healing of wounds in the stomachs of young rats was decidedly retarded by giving only one-half the required amount of an adequate diet. This retardation in healing of the wounds in the stomachs of young rats can possibly be explained by a reduction either separately or in combination of certain elements of the diet. The most probable elements are the vitamins, especially in relation to the deficiency of proteins and salts. The study demonstrated conclusively the remarkable ability of wounds of the stomachs of adult animals to heal in spite of great variations in the amount of food consumed. This ability explains the clinical success of various postoperative dietary regimens which differ in the time when feeding should begin and the amounts of food that should be given.

SPASTIC PARAPLEGIA IN ACHONDROPLASIA

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In discussing the well known pathologic entity of achondroplasia, the textbooks mention that the disturbance in growth takes place in the bones of the extremities and in those of the pelvis and the skull, and point out that the spine is usually not involved in the process. But it is known that achondroplastic infants are frequently stillborn, and in these cases the spine also may demonstrate marked, even the severest, disturbances of growth, so that the trunk and the extremities together may not be more than one and one-half times the length of the skull. In these cases there is a premature bony union between the vertebral body and the neural arch, bringing about a narrowing of the spinal canal, which is probably the cause of death in the achondroplastic fetus. The foramen occipitale magnum especially seems to be narrow, thus causing a compression of the medulla oblongata.

Generally, there is no marked difference in the length of the spine of an achondroplastic person and that of the spine of a normal person, so that when an achondroplastic person is sitting he may appear perfectly normal (*Sitzriesen*). It is known, however, especially since the investigations of Breus and Kolisko,[1] that the growth of the neural arch in the achondroplastic spine may be markedly disturbed. One sees here a narrowness of the spinal canal, especially in the frontal diameter and in the sagittal diameter. Breus and Kolisko found this to be the case. By measuring two achondroplastic skeletons, Donath and Vogl[2] ascertained that in the same spine the foramina of some segments were narrowed in the frontal direction, others in the longitudinal direction and still others in both directions, to almost the same degree.

Carrying out further investigations on achondroplasia, Donath and Vogl found that there is always a more or less marked change in the physiologic lordosis at the dorsolumbar region, and that only in this region (from the twelfth dorsal to the third lumbar segments) do the bodies of the vertebrae show a more or less pronounced wedging. In their series of seventeen adult achondroplastic dwarfs, they

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1. Breus, Carl, and Kolisko, Alexander: Die pathologischen Beckenformen, Vienna, Franz Deuticke, 1900.

2. Donath, J., and Vogl, A.: Untersuchungen über den chondrodystrophischen Zwergwuchs, Ztschr. f. d. ges. Neurol. u. Psychiat. **111**:333, 1927.

found varying degrees of kyphosis. Another seventeen showed flat backs, and only seven a distinct gibbus. In the light of these findings, the statement of Murk Jansen [3] that achondroplastic patients always have a dorsolumbar kyphosis seems to be exaggerated.

Narrowness of the spinal canal and kyphosis of the dorsolumbar region can bring about a lesion of the spinal cord, depending on the degree of development. These deformities may be so marked that life is incompatible with them, and so death occurs at birth or soon after birth. If there are, however, cord symptoms in an adult achondroplastic patient, these symptoms cannot, as already pointed out by Albrecht and Ranzi [4] and by Donath and Vogl,[2] be due to the spinal deformity alone. The following facts support this statement:

1. There are relatively few cases with lesion of the cord reported as compared to the incidence of spinal deformities.

2. In the few cases that have been reported, the symptoms appeared after bony growth of the spine had stopped. For this reason, the achondroplastic disturbance of growth itself cannot be looked on as the cause of the paraplegia. There must be something more.

CASES FROM THE LITERATURE

The following cases, which show a relationship between achondroplasia and neural symptoms, are to be found in the literature.

CASE 1 (Falta,[5] 1913).—A boy, aged 17, presented the following symptoms: ease of fatigue, paresthesia of the lower extremity, flexion contracture of the knees, spasticity and disturbed sensibility from the level of the costal margin downward. Falta ascribed these symptoms to an achondroplastic disturbance of growth.

CASE 2 (Maas,[6] 1920).—A woman, aged 71, was symptomless until the age of 61, when bladder tenesmus and difficulty in walking developed. There were loss of reflexes and hypesthesia in the lower extremities. The author does not give a complete clinical or autopsy report. The photograph of the patient's skeleton seemed to show that there was a definite decrease in the height of the first lumbar vertebra, together with marginal exostoses of the lumbar spine.

CASE 3 (Keményffi,[7] 1924.—An entire family had achondroplasia (the father and eight children). The father showed positive Babinski and Oppenheim signs in later years.

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3. Jansen, Murk: Das Wesen und das Werden der Achondroplasie, Ztschr. f. orthop. Chir. **32**:1, 1913.

4. Albrecht, O., and Ranzi, E.: Kompressionsmyelitis bei Chondrodystrophie, Wien. klin. Wchnschr. **39**:1241 (Oct. 21) 1926.

5. Falta, Wilhelm: Die Erkrankungen der Blutdrüsen, Berlin, Julius Springer, 1913.

6. Maas, O.: Beitrag zur Kenntnis des Zwergwuchses, Ztschr. f. d. ges. Neurol. u. Psychiat. **57**:196, 1920.

7. Keményffi, quoted by Donath and Vogl.[2]

CASE 4 (Albrecht and Ranzi,[4] 1926).—A man, aged 48, was symptomless until 40 years of age, when cramps slowly developed in the lower extremities and paraplegia occurred. He was unable to walk and had become bedridden for a period of one year prior to admission. From time to time he was incontinent. A diagnosis was made of transverse myelitis with a disturbance of sensibility below the first lumbar segment. Roentgenograms of the spine showed a slight kyphosis at the level of the twelfth dorsal vertebra, the height of which was somewhat diminished. The entire lumbar and lower dorsal vertebrae showed marginal exostoses, both anteriorly and laterally. Because the transverse myelitis was thought to be caused by an extramedullary factor, laminectomy of the tenth, eleventh and twelfth dorsal vertebrae was performed. The posterior arches were found to be made up of tough bone, which made their removal difficult. There was no pulsation of the dura, but it protruded markedly from the operative gap, suggesting that the spinal canal was too narrow for the dural sac. At the level of the tenth dorsal vertebra there was found a circular constriction of the dura. An incision was made through the dura below this constriction, which revealed adhesions between the dura and the leptomeninges. There was no tumor, but there was a marked softening of the spinal cord for a distance of about 1.5 cm., corresponding to the constriction. A probe was passed and met with no obstruction. The progress of the patient was remarkable, and improvement was noted on the first day after operation. The retention catheter was removed soon after the operation, and the patient subsequently walked with the aid of tripods.

CASE 5 (Donath and Vogl,[2] 1927).—A man, aged 57, had had a marked gibbus since birth. Six months prior to admission he noticed increased stiffness of the left lower extremity and difficulty in walking. He was incontinent, and tenesmus was present. Later, the right leg became involved, and he was unable to walk. The Wassermann reaction was positive. A diagnosis of a syphilitic process of the spinal cord in the region of the gibbosity was made. Antisyphilitic treatment was given, but without improvement. Laminectomy was refused by the patient. Death at a later date was due to sepsis, arising from a furuncle on the lip. Postmortem examination was performed by Professor Erdheim and showed, among other facts, the following:

To save the spinal cord for microscopic investigation and the vertebral spine for maceration, vertebra for vertebra was disconnected individually and the spinal cord isolated. All of the vertebral foramina were extremely narrow, so that the spinal cord filled the spinal canal completely. At the level of the eleventh dorsal vertebra the spinal cord was somewhat thinned out and softened. There was gray degeneration of the posterior columns; there was a severe angular kyphosis at the region of the twelfth dorsal and first and second lumbar vertebrae. All of these vertebrae were wedge-shaped, especially the first lumbar, the anterior half of which was missing. Its upper and lower surfaces formed an angle of 90 degrees. This deformity brought into contact in their anterior portions the twelfth dorsal and the second lumbar vertebrae, which were also wedge-shaped, though to a lesser degree. These three vertebral bodies showed marked marginal exostoses.

Where the spinal cord was thinned out there was not only narrowing of the intervertebral foramina, as was found in all the other segments, but there was additional narrowing due to the presence of marginal exostoses of the intervertebral joints between the tenth and eleventh dorsal vertebrae. Erdheim believes that these marked arthritic changes arise as a result of the unusually large compensatory range of motion in these joints. There was practically no motion in the area between the twelfth dorsal and the third lumbar vertebrae because of fibrous and even bony fusion of the spinous processes. The articular processes were rudimentary.

CASE 6 (Weger and Nissenbaum,[8] 1929).—A man, aged 40, had a neuropathic constitution, and had shown polyneuritic symptoms for a period of three years. The upper and lower extremities, the muscles of the face, the abdomen and the back were involved. There was marked improvement after two months of physical therapy. The authors expressed the belief that the symptoms were due to spondylarthritic changes in the lumbar spine and to the presence of bilateral rudimentary cervical ribs. This is in accord with the views held by Albrecht and Ranzi and by Donath and Vogl.

The six cases cited show a relationship between achondroplasia and neural symptoms of the lower extremities. Of these, only three (the cases reported by Falta, Albrecht and Ranzi and Donath and Vogl) can be considered as typical. The others are not sufficiently well reported to warrant any definite conclusion. The case reported by Weger and Nissenbaum obviously does not belong to this group; it certainly represents a nervous lesion of quite another character. The best contribution is undoubtedly that of Donath and Vogl, because, on the basis of Erdheim's postmortem examination, they cleared up completely the relationship between achondroplasia and the lesion of the spinal cord.

AUTHOR'S CASE

The following case is one of chondrodystrophy with paraplegia in which, as in the case of Albrecht and Ranzi, a laminectomy was performed, but unfortunately with less success.

A typical female chondrodystrophic dwarf, aged 21, with marked kyphosis at the dorsolumbar region, was admitted to the hospital in January, 1931. Since April, 1930, she had noticed an inability to lift the left foot off the ground. This symptom became more pronounced; there was marked pain in the left leg, and she noticed a tendency to twitching. On physical examination, a slight atrophy of the left lower extremity and marked spasticity, which was also present on the right side, but to a lesser degree, were found. The knee jerks were hyperactive; there was a definite ankle clonus on the left side. There was disturbance of sensibility for all stimuli. Active motion was markedly diminished on the left side, and there was some impairment on the right. There was no tenderness over the kyphosis. Good control of the bladder and bowel was maintained. These findings suggested a lesion of the third lumbar segment of the spinal cord.

Roentgenologic studies of the extremities and the skull revealed the signs typical of achondroplasia. The spine showed a mild scoliosis to the left in the lumbar region and a marked angular kyphosis of about 90 degrees in the dorsolumbar region, the apex of which was formed by the second lumbar vertebra. The body of this vertebra was distinctly smaller than the adjacent bodies, and it was definitely wedge-shaped. There was slight posterior displacement of this body. The lower anterior portion of the first and the upper anterior portion of the third lumbar vertebrae were in apposition and showed in these areas considerable osteosclerosis and marginal exostoses. A larger marginal exostosis was also seen on the superior surface of the first lumbar vertebra on the right side.

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8. Weger, A. M., and Nissenbaum, M. W.: Zur Frage der Erkrankung des Nervensystems bei Chondrodystrophie, Arch. f. Psychiat. **87**:498, 1929.

On admission, the patient was placed on a frame, and traction was applied to the head with countertraction on the lower extremities. Some improvement was noticed. The reflexes returned to normal, and the cramps in the left calf disappeared. This improvement, however, was only temporary and was followed by recurrence of all the symptoms.

On March 25, 1931, laminectomy was performed. The posterior arches of the twelfth dorsal and first and second lumbar vertebrae were removed. The cord was found to be angulated to about 70 degrees. There was no pulsation. The dura formed a thin transparent membrane which was tight over the cord. A probe passed above and below this point met with no obstacle. The dural sac was not opened.

Lateral roentgenogram, showing the wedge-shaped second lumbar vertebra with its slight posterior displacement. The marked inclination of the first and third lumbar vertebrae, with the marginal exostoses on the anterior side, is evident.

Following the operation, the patient's condition became distinctly worse, and she presented the picture of a flaccid paraplegia. There was no active motion in the lower extremities. Total anesthesia was found in the left leg and on the inner side of the right leg. At this time, the patient complained of severe cramps in both legs. Bowel and bladder symptoms were marked.

Because of these symptoms, it was thought that there might be a hematoma, and the patient was reoperated on, but no hematoma was found. One month following operation the patient showed slight improvement. There was return of some motion in the toes of the right foot and in the flexors of both knees. At a later date, some sensation in both thighs reappeared. In September, 1931, a suprapubic cystotomy was performed because of a purulent cystitis.

At the time of the last examination, in November, 1932, the patient was able to walk on crutches with the aid of two long leg braces, but only a few steps. The retention catheter was still in place.

COMMENT

This case can be classified as one of typical lesion of the cord in an achondroplastic deformity of the spine. It is difficult, however, for me to believe, as did Falta, that the stenosis of the spinal canal alone is the cause of the lesion of the cord. If Falta's supposition were true, it would be impossible for patients with achondroplasia to survive the period of bony growth of the spine.

The part which the dorsolumbar gibbosity plays in the development of neural symptoms must now be considered. The gibbosity in the case reported by Donath and Vogl, as shown in the lateral x-ray film, was essentially the same as that of my patient. However, in Donath and Vogl's case the gibbosity was at the level of the twelfth dorsal and first and second lumbar vertebrae, while in my case the deformity occupied the area of the first three lumbar vertebrae. In both of these cases the wedge-shaped vertebrae at the apex of the kyphosis showed a slight posterior displacement. As there is already a narrowing of the spinal canal and there is no further space available for the spinal cord to escape any extramedullary pressure, it would seem on first thought that this posterior displacement was the cause of the spastic paraplegia.

There is ample reason to believe that as the patient grows older, posterior displacement becomes more and more marked. Lateral x-ray films of chondrodystrophic children with a marked dorsolumbar kyphosis, as my associates and I have seen in this clinic, show deformities of the body of the vertebra at the apex of the kyphosis. But, at this stage, the vertebra still has an anterior area. On account of the high degree of kyphosis, it can be supposed that this deformed vertebra will later lose its anterior area completely and form instead an anterior edge, as seen in Donath and Vogl's and in my case. It is presumable that posterior displacement goes hand in hand with these changes through the years.

That view also must have been the one held by Donath and Vogl. Furthermore, they believed that a syphilitic process was likely to develop in this area as a locus minoris resistentiae. With this supposition of a syphilitic process, it is possible to explain the fact that the sensory changes in their patient corresponded to a lesion of the cord a segment or two above the level of the deformity in the spine itself. (The sensory changes of the body pointed toward a lesion of the second lumbar segment, while the spinal deformity was at the level of the twelfth dorsal and first lumbar vertebrae, which would mean the third and fourth lumbar segments of the cord.)

In my case, the wedge-shaped and slightly posteriorly displaced vertebra was the second lumbar. A compression caused by this vertebra would give rise to a relatively small degree of sensory disturbance, for

at this level there are, normally, the conus terminalis and the beginning of the cauda equina. But, at the time of admission, my patient already showed sensory disturbances pointing to a lesion above the level of the gibbus.

Consequently, in my case, as well as in the case reported by Donath and Vogl, the clinical analysis indicated a lesion of the cord above the level of the gibbus. Therefore, the lesion of the cord could not have been produced by the wedge-shaped vertebra. Autopsy in their case showed this to be true. There was a compression of the cord due to the projection of marginal exostoses. These marginal exostoses were found protruding from the joints of the vertebrae adjacent to and above the deformity. At this level increased motion had taken place over a long period of time as a compensation for the practically lacking motion at the level of the gibbus, and therefore hypertrophic arthritic changes developed.

Because my case resembles in almost minute detail the case reported by Donath and Vogl, it can be assumed that the same pathologic changes are present as were found in their case at autopsy. Although my patient is still only 21 years of age, there is already definite evidence of spondylarthritis deformans, especially of the vertebral bodies at the level of the deformity. In the roentgenogram arthritic changes of the small intervertebral joints are not demonstrable, but that is not remarkable as it is known that these joints are difficult to demonstrate in the x-ray film. One can, therefore, only suppose the presence of arthritic changes in these joints. One cannot prove them. It might be well to emphasize at this point that the passing of a probe without obstruction into the spinal canal at the time of operation is not evidence against the existence of exostoses. The probe was passed posteriorly. The exostoses appear laterally and anteriorly to the cord. In the case reported by Albrecht and Ranzi, too, a probe could be passed without obstruction, but they, nevertheless, thought that spondylarthritis deformans was the cause of the lesion of the cord. It is known, from recent observations, that compression of the cord can be caused by marginal exostoses occurring in spines having a canal of normal width; how much more likely is this to happen in the patient with achondroplasia who has a narrowed vertebral canal.

The question of therapeutic measures in these cases requires some discussion. In the case reported by Albrecht and Ranzi, certainly in point of deformity one of the mildest, laminectomy was followed by marked improvement of the symptoms. Although there was not complete recovery, the patient could walk again a short time ofter operation, and he regained control of the bladder and the bowels. In the case reported by Donath and Vogl, the patient refused laminectomy,

but the authors believe, because of the autopsy report, that removal of the projecting marginal exostosis would have given relief of the cord symptoms.

In my case, conservative measures were used first, such as recumbency and traction on head and legs, but without any lasting success. If one accepts as correct the mechanism of the lesion of the cord as previously explained, it is really difficult to understand how a lasting relief could be obtained by conservative means.

Because of the negative result of conservative treatment, laminectomy was finally performed, but unfortunately it did not bring any relief; on the contrary, it induced a flaccid paraplegia with paralysis of the bladder and rectum. It is not possible to give a satisfactory explanation for this fact on the basis of the operative history alone. One can understand that the laminectomy did not improve the symptoms, but why it should bring about a complete transverse myelitis is difficult to comprehend. But, since a serious traumatization of the spinal cord during laminectomy was out of the question, it may be that a minor trauma, as, for instance, sponging, produced the bad result, and that the tissue structure of the spinal cord was certainly softened at the time of operation.

With this explanation, it is still believed that laminectomy is the only therapeutic measure which, on account of the anatomic conditions, gives some hope of improvement. The sooner it can be carried out the better. It should be performed at the region of the deformity and one or two segments higher up. If there are projecting exostoses of the intervertebral joints, they must be removed, and, if possible, a fusion of these points should be performed. The dura mater should be opened in every case and should be kept open. It is not only the good result in the case reported by Albrecht and Ranzi that entitles me to this demand, but especially the result in one of Jaroschy's[9] cases of scoliosis with compression of the spinal cord (these cases of scoliosis are in close relationship to cases of chondrodystrophy). Jaroschy performed in his case, first a simple laminectomy without opening the dura; there was no relief from symptoms after the operation. He therefore reoperated, opening the dura, and the patient showed good recovery from the symptoms.

Finally, one has to consider whether some prophylactic measure could be carried out in cases of achondroplasia with severe dorsolumbar kyphosis. As mentioned, it is to be taken for granted that there is a gradual increase of deformity with the years. The secondary, marked, arthritic changes which may occur in early years are undoubtedly a

9. Jaroschy, W.: Ueber Spätschädigung des Rückenmarks bei kongenitaler Skoliose und ihre operative Behandlung, Beitr. z. klin. Chir. **129**:348, 1923.

consequence of the abnormal static and mechanical conditions of the spine. In all cases of marked kyphosis in which an increase of deformity can be expected later on, with a possible lesion of the cord, it seems logical to perform a preventive spinal fusion at an early age, if possible in childhood, when most of the chondrodystrophic dwarfs come to medical observation. This fusion might be performed at the whole dorsolumbar region after the method of Hibbs. Removal of the posterior arches of the segments which form the deformity might also be taken in consideration. But whether there is indeed a general indication for this operation, as in some cases of scoliosis, can be established only by careful statistical examination. If these statistics prove that cord symptoms develop later in a high percentage of cases of chondrodystrophy with marked dorsolumbar kyphosis, then this operation would seem to be indicated.

INTUSSUSCEPTION ASSOCIATED WITH TUBERCULOSIS

A CASE IN AN ADULT

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The following case of enteric intussusception which recently came under my observation presented some puzzling features, both as to immediate cause and ultimate result:

History.—R. O., a man, aged 32, was admitted to Knickerbocker Hospital on April 30, 1932. He was very emaciated and acutely ill. The onset of illness was two days previously, when he suddenly felt a dull pain in the region of the umbilicus. Half an hour later he vomited mucous material, and within a few hours some bright red blood. After that, the vomitus was black. There was no bowel movement for two days. Numerous enemas brought no results. The abdominal pain became sharp after his admission to the hospital. The abdomen was slightly distended, with a boardlike rigidity. No peristalsis was visible. There was tenderness just below the umbilicus, where an indefinite mass was felt. Palpation of the abdomen induced peristalsis and cramps.

The patient gave a history of good health up to the onset of the present illness, except for a bilateral inguinal hernia of some years' duration.

The heart was normal, the lungs clear, and the temperature 100 F.

The symptoms pointed to intestinal obstruction, probably in the lower portion of the ileum, and immediate operation was imperative.

Operation and Course.—A right rectus incision was made 6 inches (15.2 cm.) long with the center opposite the umbilicus. The stomach, gallbladder and appendix were normal. There was a 4 inch (10.2 cm.) intussusception of the ileum, 12 inches (30.5 cm.) above the ileocecal junction. The intestine was collapsed below this point and distended above it. This intussusception was reduced by gentle expression and traction, disclosing several bluish and hemorrhagic areas, which were pregangrenous. A search for polypi or other tumors which might account for the intussusception was unavailing. The mesentery, however, was unusually long and relaxed and showed a marked absorption of adipose tissue. It gave the impression of a state of extreme ptosis. The mesenteric vessels appeared to have good circulation, and nothing of an unusual nature was discovered except the slight bluish and pallid indurated areas of the intussusceptum, one of which was enfolded with serous suture. There was considerable serosanguineous free fluid in the abdomen.

After reduction of the intussusception, the area was wrapped in hot packs and an enterostomy done about 2 feet above it in the distended portion of the intestine. On reexamination, the condition of the intestine as a whole seemed to have improved greatly since the reduction. A rubber dam drain was placed in the area of the enterostomy, and the abdomen was closed in layers.

Three days after operation the drainage had become profuse, and the drain was removed. The ileostomy was in good condition. The bowels moved only with enemas.

On the following day, the ileostomy tube, which had become loose in the wound, was removed, and the intestinal contents drained freely through the wound. At this time the sutures were cutting into the skin and were removed.

Seven days after operation, the patient was removed to a private room for suction application to the wound. However, this blocked up frequently and allowed the intestinal contents to irritate the edges of the skin. Bronzing liquid and powder were used in combination to prevent this irritation. A collapsible rubber ball and iodoform packing were placed in the sinus, but as this did not prevent leakage, gauze treated with petrolatum was substituted. Every effort to preserve the skin at the edges of the wound proved unsuccessful, and a side-to-side anastomosis was considered, but was temporarily postponed.

A month after operation the patient was losing weight; barium injected through the fistula showed no obstruction, and feeding through the intestinal opening was suggested. At this time there was no evidence of a pathologic process in the lungs except cough, which was thought to be due to excessive smoking.

During the next four weeks the patient continued to lose weight and strength. Blood transfusions were given. On June 29, the abdomen was reopened by Dr. J. V. Bohrer for the repair of the intestinal fistula.

A low, left pararectus incision was made downward 5 inches (12.7 cm.) from the umbilicus. Numerous adhesions had formed, which were easily separated. There was no evidence of the previous intussusception. The fistula was separated from the anterior abdominal wall, and the hole in the intestine was closed with catgut. An entero-enterostomy was then done, short-circuiting the newly closed fistula. This portion of the intestine was lightly attached to the anterior abdominal wall near the former fistula.

On the third postoperative day the wound showed no evident healing. The skin sutures were cutting. The patient vomited a dark, brown fecal-odored material. A Levine tube was inserted to the duodenum; during this manipulation the patient vomited 2,000 cc. of this brown liquid. An enema yielded a liquid return of a brownish color.

A furuncle on the left wrist was incised and drained.

The general condition of the patient became gradually worse, with death on the sixth postoperative day.

Autopsy.—Considering the patient's apparently healthy condition prior to the acute obstruction which brought him to the hospital, it was difficult to determine the cause of the intussusception and his lack of resistance to the operation. Fortunately, an autopsy was permitted, which threw some light on the matter, and yet leaves a satisfactory explanation still in the field of conjecture.

A partial report of the examination follows:

The lungs showed chronic, active, bilateral, apical tuberculosis, with bilateral, caseating tuberculous bronchopneumonia in both lower lobes, and tuberculous pneumonia in the left upper lobe.

Parenchymatous degeneration was seen in the liver and kidneys.

Examination of the intestinal tract showed necrosis of the ileum in the region of a recent ileostomy; chronic localized peritonitis of the terminal ileum and ascending colon; ulcerative tuberculous colitis; diverticula of the duodenum, and active tuberculosis of the mesenteric lymph nodes.

The blood vessels of the mesentery were in good condition, but there were marked sclerosis and contraction of the mesentery of the lower portion of the ileum such as occur in cases of old gunshot wounds.

Section through the small intestine disclosed the presence of epithelioid tubercles in the mucosa.

Section through the cecum revealed a small, shallow ulcer of definite tubercle formation, with giant cells in the deeper layers of the mucosa and submucosa.

The report leaves no doubt as to the immediate cause of death, which is stated to be "tuberculous bronchopneumonia, localized peritonitis, necrosis of the ileum and old intussusception." The direct cause of the intussusception, however, still remains in doubt, since there was no neoplasm or tuberculous ulceration at the site of the lesion.

ETIOLOGY

An extensive survey of the literature of both tuberculosis and intussusception with their complications has failed to reveal a case similar to the one under discussion. Eliot and Corscaden [1] made a survey of the whole subject of intussusception in adults in 1910, citing 300 cases, in only 10 of which the disease was associated with tuberculosis with ulcer formation or serous or subserous infiltration. In the case herewith reported, the single tuberculous ulceration was confined to the cecum, while the invagination was located in a portion of the ileum a foot from the ileocecal valve and showed no evidence of ulceration. Although a section in this area disclosed epithelioid tubercles in the mucosa, it is highly improbable that this caused the intussusception.

Correspondence with the surgeons particularly concerned in handling cases of tuberculosis at the nearby hospitals brought to light no instances of intussusception in recent years associated with tuberculosis such as the one herewith reported or those reported by Eliot and Corscaden.

Rubin [2] collected the reports of 500 autopsies on patients who had died of pulmonary tuberculosis, viewed with special reference to the condition of the intestines. Macroscopic ulcerations were revealed in the intestines in 324 of the 500 cases, ranging from a few incipient lesions to an extensive involvement of the whole intestinal tract. Yet, there is no mention of an intestinal intussusception in the whole list.

In view of these facts, pulmonary tuberculosis as a direct cause of intussusception is difficult to establish. But there is no doubt that it constitutes a predisposing factor. Gastro-intestinal disturbances have been noted by various observers in from 70 to 92 per cent of the cases of early tuberculosis. In the acute forms they are always present to a marked degree. As the disease progresses and emaciation becomes more and more pronounced, evidence of enteroptosis with

1. Eliot, Ellsworth, and Corscaden, James A.: Intussusception with Special Reference to Adults, Ann. Surg. **8**:169 (Feb.) 1911.

2. Rubin, E. H.: Pulmonary and Secondary Intestinal Tuberculosis, Am. Rev. Tuberc. **22**:184 (Aug.) 1930.

atony of the stomach is increasingly seen. In cases of moderate severity, in which there is little or no secondary invasion of the gastro-intestinal tract by the disease, the abdomen is unusually soft, relaxed and extremely tender. A gastric catarrh is by this time almost always present. The gastric contents show a high degree of both motor and secretory insufficiency, the gastric acidity being greatly reduced. The bowels also are commonly affected, first by constipation and later by occasional severe attacks of diarrhea which increase in frequency and cause intense weakness and exhaustion.

Thus there is a condition of lessened resistance throughout the gastro-intestinal tract, inviting disaster, as it were, from the impact of any unaccustomed shock or strain to which it may be subjected.

Among the direct causes of intussusception in the adult, in the small intestine, by far the most common is the presence of a polyp, benign tumor or foreign body; in the large intestine, on the contrary, it is usually a malignant tumor. The neoplasms are usually found at the apex of the intussusceptum and are pedunculated in form.

Ulcers arising from typhoid fever or intestinal tuberculosis are frequent causes, as are paralytic conditions of the intestine which allow the collapse of one portion into another.

Trauma is considered by some authorities an important causative factor. Strong movements of the muscles as in severe coughing, falls from a height, heavy lifting and football playing are given as instances.

Diet appears to play an important part. Orr [3] cited the case of an Indian coolie 30 years old, very thin and poorly nourished, who came to the hospital with an enteric intussusception involving 3 feet of the ileum. After resection and side-to-side anastomosis, a second large intussusception formed in a proximal portion of the intestine. Even after this was reduced there were violent and irregular peristaltic movements passing along the intestine as if another intussusception were attempting to form. In the absence of anything in the abdomen to suggest a cause for the intussusception, it was attributed to the patient's defective diet, which had consisted of tapioca, rice water, tamarind and red chillies, with occasional small quantities of fish.

Contrary to the rule elsewhere, in India there seems to be a predominance of cases in adults over those in infants. In one hospital 66 per cent of the cases occurred in persons over 20 years old. In only two of these was there any record of a polyp or structural defect. Some upset in the neuromuscular control of the bowel, probably due to faulty diet, seemed indicated in the rest.

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3. Orr, Ian M.: An Unusual Case of Intussusception in an Adult, Brit. M. J. **1**:331 (Feb. 20) 1932.

The direct effect of an improper food balance was shown by McCarrison in his experiments on monkeys. These were put on a regimen deficient in vitamins, low in proteins and high in carbohydrates. Among other changes in the gastro-intestinal tract, a very high percentage of intussusception was noted. From this, Dr. McCarrison [4] traced a definite causative relationship between dietetic insufficiency and intussusception. "No doubt there is nothing new in such a statement," he observed, "but I do not think we have realized hitherto that the neuro-muscular control of the bowel is dependent in great measure on the adequate provision of vitamins in the food."

The use of castor oil as a household remedy is held by some authorities to be largely productive of intussusception, owing to the strong, irregular peristalsis it induces. Kock and Orum [5] believed this to be the reason for the numerous cases found in Great Britain and Denmark as compared with France and Germany, where it is less widely used.

Owing to the difference in diameter of the small and the large intestine, the ileocecal or ileocolic type of intussusception is by far the most frequent. In a wide survey, Perrin and Lindsay [6] found the ratio between this and the enteric type to be about 6:1. In the absence of definite etiologic factors, they considered the cause of enteric intussusception probably to be found in the swelling of a Peyer's patch. Briefly formulated, their theory to account for a majority of primary intussusceptions is the production of the equivalent of a foreign body within the wall of the intestine. This foreign body is provided by the swelling of a preexisting lymphoid tissue. The anatomic and age distribution of the lymphoid tissue in the alimentary canal agrees exactly with the anatomic and age distribution of all primary intussusceptions. Hence children are more liable. The factor that provokes this swelling is usually some gastro-intestinal disturbance, with resulting chemical irritation; the formation of gas from indigestible foods, such as cabbage and sausage, may be sufficient to cause it.

Another possible cause of intussusception is tuberculosis of the mesenteric lymph glands. This condition is a definite clinical entity. It is found to occur most commonly between the ages of 20 and 30. The patients are, as a rule, not of the rugged type, but rather slender, poorly nourished and anemic; they tire easily, have lost weight or fail to gain in weight.

Despite its frequency, little attention has been directed to this disease, and its surgical significance is seldom recognized in connection with abdominal operations.

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4. McCarrison, quoted by Orr.[3]

5. Koch, A., and Orum, H. P. T.: Bibliot. f. læger **12**:333, 1911.

6. Perrin, W. S., and Lindsay, E. C.: Intussusception, Brit. J. Surg. **9**:46 (July) 1921.

The enlargement of the mesenteric glands, through this infection or otherwise, is recognized as a cause of various gastrointestinal symptoms, among which are pain, tenderness, rigidity and perhaps a slight rise in temperature; also colicky dysfunctions of the digestive tract, pyloric spasm, spasticity of the abdominal muscles and nervous irritability, which probably arise reflexly from irritation of the autonomous nerves by the enlarged glands.

Alvarez [7] stated that it seems probable that many of the strange disorders of digestion associated with slight fever, nausea, abdominal soreness, signs of reverse peristalsis and diarrhea could best be explained on the basis of such a low grade tuberculous infection of the lymph nodes. In his opinion, one of the greatest needs of medicine today is some means of raising the resistance of the body to such infections. He suggests that symptoms of this sort be treated exactly as if they were known to be of tuberculous origin, i. e., by rest, overfeeding and heliotherapy.

An enlarged tuberculous lymph gland can directly produce intussusception by pressure into the wall of the intestine, forming a pocket which grows gradually deeper under the peristaltic movements of the bowel until there is a complete invagination, with the lymph node enfolded at the apex of the intussusceptum. This process has been graphically shown by Lorenz [8] in the case of a small mesenteric cyst which, pressing into the ileum, forced its way through the ileocecal valve and produced a deep ileocecal intussusception.

Sherman [9] reported the case of a child showing every symptom of intussusception. When the abdomen was opened, a much enlarged tuberculous lymph gland was found lying close enough to the spine to press against the mesenteric veins and partially occlude them. The intestine served by these obstructed veins was thick, edematous and congested. While no intussusception was found in this case, it may be said that the stage was all set for such a catastrophe, which doubtless would have occurred had not resection of the affected portion of the intestine with its mesentery been promptly done.

All stages of tuberculosis may be found in the mesenteric lymph glands—tubercles, caseation, liquefaction and all degrees of calcification.

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7. Alvarez, Walter C.: Mesenteric Lymphadenitis in Adults, Cause of Pseudo-Appendicitis, Indigestion, Diarrhea and Arthritis, M. Clin. North America **14**:605 (Nov.) 1930.

8. Lorenz, Hans: Ein Beitrag zur Lehre von der Invagination, Deutsche Ztschr. f. Chir. **77**:7, 1905.

9. Sherman, Harry M.: Tuberculosis of Mesenteric Lymph Glands, Symptoms of Intussusception Necessitating Resection of the Intestine, California State J. Med. **4**:43, 1906.

According to Ross Golden,[10] the complications which may follow are: (1) adhesions from the nodes to the intestine or to the abdominal wall, which may produce ileus; (2) pressure on other structures, such as the stomach or the ureter; (3) perforation of a caseous node into the peritoneal cavity, and (4) generalized miliary tuberculosis. The symptoms of these may entirely mask the original disease. While Golden makes no mention of intussusception as a sequel, its probability may legitimately be inferred.

Tuberculosis may exist in the mesenteric lymph glands without tuberculous involvement of other organs. In the gastro-intestinal tract bacilli little affected by the gastric juices pass readily through the stomach, where little absorption takes place, with no apparent weakening of their virulence. They pass rapidly through the duodenum and jejunum, where the food, mixing with digesting fluid, is being prepared for use. In the ileum the first effective absorption of the contents of the alimentary canal naturally occurs. There, as in the case of typhoid bacilli, tubercle bacilli pass the epithelial barrier to the submucosa and act on the solitary follicles or Peyer's patches, and make tubercles in the mesenteric nodes. Brown and Sampson [11] have shown little mammilations giving evidence of healed lesser grades of tuberculous infection of the lymphoid structures of the submucosa of the small intestine. Auchincloss [12] stated that he has often looked for gross evidence of scarring in the intestinal walls, but found none. It is probable that most cases of tubercle bacillus infection of the mesenteric glands show no evidence of disease in the intestinal wall.

In an adult, these diseased glands may be the cause of ill health existing from childhood. On the other hand, in many cases clinical symptoms do not develop and may be discovered only when the patient is being examined for another disease. Such lesions seems to indicate a handicapped area in one of the most important functioning sections of the body, and when the individual as a whole, or the area in particular, is subjected to excessive stress or activity or infection, it will not function; it breaks down, and so-called intestinal symptoms appear.

The lymph and lacteal drainage for the corresponding part of the intestine has been diseased beyond repair. The node has been walled off as a foreign body as nearly as possible. A rearrangement and compensatory mechanism have had to be established, the efficiency of which is

10. Golden, Ross, and Reeves, R. J.: Significance of Calcified Abdominal Lymph Nodes, Am. J. Roentgenol. **22**:305 (Oct.) 1929.

11. Brown, Lawrason, and Sampson, H. L.: Intestinal Tuberculosis, ed. 2, Philadelphia, Lea & Febiger, 1930.

12. Auchincloss, Hugh: A Clinical Study of Calcified Nodes in the Mesentery, Ann. Surg. **91**:401 (March) 1930.

generally less than that of the original arrangement. The same is true of the blood vessels to and from the lymph nodes and adjacent to them. Damage may have occurred to the sympathetic nerve trunks in the region, impairing their nutrition and giving rise to symptoms that may be spastic or paralytic.

The facts here presented afford a framework into which the clinical picture of the case under discussion may be found roughly to fit.

A weak, undernourished man suffered from active tuberculosis of the lungs, which alone would tend to lower the resistance of the gastro-intestinal tract, as has been shown, and make him liable to serious consequences under any unusual stress or strain. The enlarged tuberculous mesenteric lymph glands were capable of causing invagination by pressure into the intestinal wall, or by pressure on the sympathetic nerve trunks producing paralysis of the intestine. His mesentery was long and markedly lacking in fat, and the stomach and intestines probably atonic and ptosed—a condition peculiarly favorable to ileus, either through volvulus or intussusception. The question is, what took place in this area in which every preparation seems to have been made to bring about the final catastrophe—intussusception?

It may have been, as Perrin and Lindsay suggest, a swelling of the Peyer's patches which acted as a foreign body. In the light, however, of new facts in the patient's history which have just come into my possession through the kindness of his widow, I believe the determining cause more likely to have been trauma.

The patient was a waterproofer by trade, and had returned to his work just two days before the onset of his illness, after driving a taxicab for a year. He was a small man averaging 126 pounds (57.2 Kg.) in weight, and "soft" when he returned to this job. Far from being in good health, as he stated, he had coughed a great deal during the previous winter, especially in connection with heavy smoking, was constipated, and had night sweats.

His appetite was good, in fact, he was "always eating something." Certain foods, however, made him vomit.

Three or four days before the onset of his illness, he ate some cabbage, which upset him as it always did.

The patient was in a weakened condition when he took up his work as waterproofer. His duties required him to lift heavy pails of tar weighing about 75 pounds (34 Kg.), one in each hand. On April 28, just two days before he came to the hospital, he almost fainted from this exertion and experienced a sudden pain in the abdomen. He was so ill he had to be helped off the job and taken to a first aid station, where he was given some paregoric. After eating sausage for lunch, he felt very sick and took some castor oil and magnesium sulphate. He did not use castor oil habitually, but relieved his constipation with a patented preparation of phenolphthalein.

After taking the castor oil and magnesium sulphate, the patient began vomiting red blood, and summoned a doctor, who, believing the symptoms to be those of gastric ulcer, treated him accordingly. Two days later he came to the hospital and the intussusception was discovered.

The fact that his illness came on in the act of lifting a heavy weight makes it appear that this unaccustomed strain was the final, direct cause of the intussusception. Eliot reports in his series seven such instances.

In view of the patient's general pathologic condition, the fatal outcome was perhaps not surprising. It may be open to question whether immediate resection and anastomosis would have shown better results.

Oschner [13] observed that the treatment in any case of acute obstruction depends on the amount of distention in the afferent loops, the degree of interference with the vascular supply of the part, and the condition of the patient. In the presence of marked distention of the intestine proximal to the obstruction, no attempt should be made to remove the pathologic process producing obstruction, since the contents of the intestine are extremely toxic and contain highly virulent organisms. The distended intestine is thinned and very friable. Resection of the pathologic process with reconstruction of the intestinal tract is not feasible. The sutures introduced into the distended pathologic wall of the bowel are insecure. The operative procedure requires more time than is warranted by the patient's condition. Any operative procedure should involve merely the relief of the obstruction. At a later date, as soon as the patient has recovered from the toxemia and signs of acute obstruction, the cause may be removed. The obstruction is best relieved by production of a fistula either by enterostomy or colostomy. This should be made immediately above the obstruction.

There is probably no one condition the surgeon encounters which causes him more concern than certain cases of ileus. Even in spite of the most careful and exacting therapy the condition often progresses to a fatal termination.

The mortality in acute obstruction is high. Van Beuren and Smith,[14] in a series of 349 collected cases, demonstrated an average mortality of 60.7 per cent after enterostomy. Wichmann [15] stated that in 223 cases of intussusception, nearly 70 per cent of the patients died after operation. Perrin and Lindsay, however, place the mortality rate much lower, their average in 400 cases being given as 34.7 per cent.

In discussing the cause of death from high obstruction, Hartwell and Cooper [16] said:

It is apparent that the cause of death following intestinal obstruction is the absorption of toxic substances into the blood. The source and nature of these substances are not fully established, nor is it certain what rôle is played by the loss

13. Oschner, Alton, in Nelson Loose-Leaf Living Surgery, New York, T. Nelson & Sons, 1927, vol. 5, p. 266.

14. Van Beuren, F. T., and Smith, B. C.: Status of Enterostomy in Treatment of Acute Ileus, Arch. Surg. **15**:288 (Aug.) 1927.

15. Wichmann: Deutsche Ztschr. f. Chir. **77**:1, 1905.

16. Hartwell, John A., and Cooper, Henry S. F., in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1929, vol. 7.

of substances, particularly water and chlorides, in the vomitus, but this is doubtless an important factor.

What is the cause of death in high obstruction? No theory of toxemia has given rise to any practical means of preventing death after the condition has fully established itself. Surgical intervention is the best means, and only too often this is of no avail. We do not know the cause of death from high obstruction. It is to be hoped that somebody will find the answer and bring light where there is now darkness.

SUMMARY

Intussusception is much less frequent in adults than in children. Approximately twice as many males as females are affected by it.

The mortality is high, being in direct ratio to the length of time after the appearance of symptoms that operation is done. There is a decided tendency to recurrence even after resection of the portion of bowel originally affected. Spontaneous recovery sometimes takes place, the necrotic intussusception becoming detached and passing through the bowels.

The disease constitutes a grave threat among persons suffering from other diseases, notably typhoid fever and tuberculosis. It is induced by such conditions as undernourishment, radical changes of diet creating hyperperistalsis, visceroptosis, mesenteric lymphadenitis, and tumors, cysts, ulcers and adhesions in the gastro-intestinal tract, while trauma undoubtedly plays an important part in many cases as an activating agent.

In cases of pulmonary tuberculosis it is a complication to be feared, since it may not only lessen the possibility of recovery by interfering with the patient's nutrition, but in its acute form is more than likely to precipitate a fatal termination. On the other hand, pulmonary tuberculosis, with its weakening effect on the gastro-intestinal tract, predisposes the patient to intussusception.

Undernourishment, through insufficient or ill balanced diet, is a definite causative factor, and perhaps the only one through which effective preventive action may be taken.

It is suggested that every tuberculous patient be regarded as a potential victim of intussusception, and his treatment planned with this complication in view. This may mean simply an intensification of the regular treatment for tuberculosis, with special emphasis on overnutrition, protection from overexertion and an increased watchfulness for gastro-intestinal symptoms.

DIAGNOSIS AND TREATMENT OF FRACTURED SKULLS

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A newly awakened and much needed scientific interest in the study of fractured skulls and intracranial injuries is evidenced by the rapid increase in the literature on this subject in the past five years. It is clear to us that there are many imperfections in our knowledge of the basic pathologic and physiologic changes that are present in these cases, and only after these gaps in our knowledge have been filled shall we be able to interpret correctly many of the clinical phenomena observed daily in the wards. A much better understanding and correlation of fundamental facts are needed to put us on the right track. Each writer has his own classification, which varies with his concepts, and this results in much confusion and controversy and renders comparative work difficult, because one is never quite sure that one author is writing about exactly the same thing that another author has described. Many of the differences are more apparent than real and are due to inability to understand one another, while, on the other hand, some are definite disagreements in fundamental ideas. It is our purpose in this paper not to offer a solution to these problems, but to report our experiences in the study and management of these cases at Harlem Hospital, and to draw certain seemingly justified conclusions therefrom. This work is supplemental to and a continuation of the studies made at Harlem Hospital by Dr. John F. Connors,[1] surgical director, which were reported by him in two separate papers, based on a study of a total of 833 cases. In our present work we were fortunate in having the benefit of his advice and supervision.

We studied 347 consecutive cases of cranial and intracranial injuries, as the patients were admitted to the wards, from Sept. 1, 1930, to April

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Presented before the Joint Meeting of the Boston, New York and Philadelphia Regional Fractures Committee of the American College of Surgeons, New York, May 19, 1932.

1. Connors, John F.: Management of Intracranial Injuries With or Without Fracture, Ann. Surg. **81**:901, 1925; The Treatment of Fractures of the Skull, Tr. Am. S. A. **14**:427, 1927.

20, 1932. From Sept. 1, 1930, to Oct. 1, 1931, we diagnosed all severe craniocerebral injuries as "clinical fractured skull," which is the classification in general use today, but the conviction gradually grew on us that only cases in which we could actually prove the presence of a fracture or fractures by roentgen examination or autopsy should be so diagnosed or classified; therefore, beginning on Oct. 1, 1931, we divided the lesions into two groups according to whether or not a fracture of the skull had been demonstrated: (1) proved fractures of the skull and (2) proved intracranial injuries without fractured skull. It is common knowledge that the treatment of fractured skulls is concerned chiefly with the treatment of the concomitant underlying injury to the brain or its coverings. We were careful to exclude all cases with questionable or only suggestive roentgenograms from our group of proved fractures of the skull. When there was a history of trauma, blood in the spinal fluid was the basis for making a diagnosis of intracranial injury in patients who lived, and of course for an autopsy in

TABLE 1.—*Classification of Cases*

	Number of Cases	Lived	Died	Mortality, per Cent
Class A. (Sept. 1, 1930, to Oct. 1, 1931)				
Clinical fractures of the skull; includes intracranial injuries	129	99	30	23.2
Class B. (Oct. 1, 1931, to April 20, 1932)				
Group 1. Proved fractures of the skull	133	98	35	26.3
Group 2. Proved intracranial injuries	85	77	8	9.4
Total	347	274	73	21.0

fatal cases. Blood in the spinal fluid owing to a ruptured aneurysm of one of the vessels of the circle of Willis occurred in rare instances (an average of about 1 such case a year). Before we made a clinical diagnosis of intracranial injury without skull fracture, we satisfied ourselves that the blood in the spinal fluid was not due to contamination resulting from the lumbar puncture. We excluded from the group of those with intracranial injury without fracture of the skull 53 patients who had been sent to the wards with the diagnosis of "possible fractured skull" or "head injury." A history of trauma was given by most of them, and a history of unconsciousness by all of them; but the results of roentgen examination were negative, and the spinal fluid was devoid of red blood cells. We realize that we may have missed 1 or 2 cases of slight cerebral injury by insisting on the presence of blood in the spinal fluid before permitting this diagnosis to be made. None of the innumerable patients with simple laceration, contusion and hematoma of the scalp who come to the hospital were included in this study, as they were treated in the emergency room of the hospital and sent home. All cases in this series were grouped as shown in table 1.

It is seen that the incidence of actual fractured skulls is much higher than the incidence of intracranial injuries without fractured skull and, too, that the mortality from intracranial injuries without fracture of the skull is only about one-third the mortality from real fractured skull.

A clinical diagnosis of fractured skull should not be made unless the existence of the fracture is proved by roentgen examination or the fracture is actually seen, as is possible in certain compound fractures or at operation. A clinical diagnosis of intracranial injury should be made only after red blood cells are found in the spinal fluid. Erythrocytes in the spinal fluid constitute the chief tangible and the most trustworthy evidence of injury that is available at the present time, and the tendency to accept this view is shown by the fact that Kennedy and Wortis [2] in a recent review of 1,000 cases of "head injuries" used the same criteria. The term "head injury" should be discarded as it is too indefinite, and although what it means today is known, it could properly include traumatisms of the face and scalp. Either there is a fracture of the skull or there is not, and either there is intracranial injury without fracture or there is not; and both conditions exhibit pathologic changes that are demonstrable clinically.

Acute alcoholism coexists so frequently with injuries of this type and in so many instances obscures the clinical picture irrespective of the degree of trauma that it becomes a factor of no inconsiderable importance. Vance [3] found alcohol in the liver and brain in 512 fatal cases. It was noted by McCreery and Berry [4] in their clinical study of 520 cases, but they could not estimate its importance. In our group of cases, a diagnosis of acute alcoholism as an associated condition was made in 54 instances, or 18.8 per cent, and we feel that the percentage is much too low, because in many of the unconscious patients the effects had worn off before we saw the patients or were masked by the concomitant injury. Alcohol can and does produce disorders of consciousness, as well as retrograde amnesia; and these two symptoms, particularly the loss of consciousness following cephalic trauma, are the most important of those produced by acute craniocerebral injury. In fact, the diagnosis of cerebral concussion is based entirely on the history, or on the presence, of loss of consciousness following trauma. We are of the opinion that alcohol plays a much larger rôle in these cases than is generally thought, and that it accelerates death in many instances, owing to its late depressant effects on all body functions.

2. Kennedy, Foster, and Wortis, S. Bernard: Head Injuries, J. A. M. A. **98**:1352 (April 16) 1932.

3. Vance, B. M.: Fractures of the Skull, Arch. Surg. **14**:1023 (May) 1927.

4. McCreery, John A., and Berry, Frank B.: A Study of 520 Cases of Fractures of the Skull, Ann. Surg. **88**:890, 1928.

Much further work is necessary to determine exactly the part that alcohol plays in these cases.

The nature of the trauma is shown in table 2. Gunshot wounds ordinarily would fall into the category of assaults, but it seemed desirable to group them separately. In the group with the "history of injury unknown" the lack of the history may be explained by death without recovery of consciousness, alcohol and retrograde amnesia. Many of these patients were found unconscious in hallways or areaways; and in not a few cases patients brought to the hospital died, and we were not able to identify them. Today the automobile is the leading causative factor in the production of fractured skulls.

As regards sex, the ratio of males to females in this series was about 3 to 1.

There were 28 children under 10 years of age, and 1, or 3.5 per cent, died. The patient that died was an infant of 2 months who was struck with a bottle and sustained a fracture involving the right frontal sinus; death occurred four days later as a result of meningitis. Our

TABLE 2.—*Nature of the Trauma in Intracranial Injuries*

	Our Cases	Connors' Cases
Automobile	120	115
History of injury unknown	87	49
Falls	77	133
Assaults	55	27
Gunshot wounds	3	0
Street cars	3	17
Subway cars	2	5

observations in general confirm the conclusions of Beekman,[5] Ireland,[6] Wakeley[7] and others relative to the better prognosis for life in craniocerebral traumatisms in children.

The time elapsing between admission to the hospital and death in the 73 fatal cases is shown in table 3.

It is seen that 38 patients, or 52 per cent, died within the first twelve hours. Fay[8] reported that 12 of 39 deaths occurred within three hours after admission to the hospital in one of his series, while McCreery and Berry recorded that 110 of 204 deaths occurred within the first twelve hours after admission. Death after forty-eight hours in most instances was due, not to laceration of the brain, but rather to a complication, such as meningitis, hypostatic pneumonia, associated injury, or exhaus-

5. Beekman, Fenwick: Head Injuries in Children, Ann. Surg. **87**:355, 1928.

6. Ireland, Jay: Fracture of the Skull in Children, Arch. Surg. **24**:24 (Jan.) 1932.

7. Wakeley, Cecil P. G.: Fractures of the Skull in Children, Practitioner **127**:75 (July) 1931.

8. Fay, Temple: Clinical Considerations Surrounding Head Injuries, S. Clin. North America **11**:1375 (Dec.) 1931.

tion and inanition. This was first pointed out by Connors, and the chief exception to this rule has been extradural hemorrhage. Death due to laceration of the brain and subdural hemorrhage usually occurs within forty-eight hours after the injury, and if death does occur later it is the exception to the rule, as happened in a case in our series.

Cerebral concussion is the term used to denote the unconsciousness that occurs after an injury to the head, and is not usually associated with fracture of the skull, in the clinical acceptation of the word. There is much controversy among observers in explaining exactly what happens and whether or not it has a pathologic basis in fact. Vance listed 27.4 per cent of the deaths in his series as due to concussion, and in some he could find no pathologic basis; all of his patients died within a few minutes or hours after the injury. Ochsner [9] expressed

TABLE 3.—*Time Between Admission to the Hospital and Death*

Time in Hospital Before Death	Class A	Class B	
	Clinical Skull Fractures	Group 1 Skull Fractures	Group 2 Intracranial Fractures
1 hour	1	8	2
3 hours	2	4	1
6 hours	5	3	1
12 hours	6	5	0
24 hours	3	4	0
2 days	4	2	0
3 days	2	2	1
4 days	4	4	1
5 days	1	0	1
6 days	0	1	0
8 days	0	1	0
9 days	1	0	0
12 days	1	0	0
14 days	0	1	0
20 days	0	0	1
Total number of cases	30	35	8

the belief that concussion is "a physiologic and not an anatomic lesion," while Riddoch [10] stated that it "may produce death presumably from bulbar anemia." Ritter and Strebel [11] described concussion of the medulla and concussion of the cerebrum. On the other hand, Rand and Courville [12] found edema of the choroid plexus in 2 cases. Peet [13] thinks that there are probably minute lacerations of the brain substance.

9. Ochsner, Alton: The Diagnosis and Treatment of Acute Craniocerebral Injuries, Am. J. Surg. **12**:222 (May) 1931.

10. Riddoch, George: Discussion on the Diagnosis and Treatment of Acute Head Injuries, Proc. Roy. Soc. Med. **25**:735 (March) 1932.

11. Ritter, A., and Strebel, K., quoted by Ochsner.[9]

12. Rand, Carl W., and Courville, Cyril B.: Histologic Studies in Cases of Fatal Injury to the Head, Arch. Surg. **23**:357 (Sept.) 1932.

13. Peet, Max Minor: Symptoms, Diagnosis and Treatment of Acute Cranial and Intracranial Injuries, New York State J. Med. **28**:555 (May 15) 1928.

Dr. C. S. B. Cassasa,[14] one of our associates, in 1925 found minute hemorrhages into the perivascular lymph spaces in 5 cases that exhibited clear spinal fluid. Kaufmann[15] stated: "That gross damage is done, such as multiple hemorrhages (tearing of vessels through shock or the push the brain substance resists), is shown by the fact that foci of white softening appear as an expression of the microscopic damage to the nervous tissue, to which may be added petechial hemorrhages." Jefferson[16] expressed the belief "that in all head injuries serious enough to call for treatment during the acute stage, there is a fundamental state due to general cerebral contusion and that this has a recognizable histological picture." He has also observed the minute hemorrhages first described by Cassasa. He would discard the use of the term concussion, and we agree with him that its use adds confusion; we also agree with him that any patient who on admission to the hospital is unconscious may be safely assumed to have definitely widespread neural damage, although we qualify the latter statement by specifying that the unconsciousness must be due to trauma and not to alcohol or drugs. It seems clear that Cassasa, Jefferson and Kaufmann have described the same pathologic condition under different names—they are all agreed that unconsciousness of sufficient degree and duration to require hospitalization has associated pathologic changes in the central nervous system, whether the latter are demonstrable or not, and it is with this idea that we are in accord. If the term concussion is to continue in use, the description of the clinical features as outlined by Trotter[17] should be used, which is as follows: (1) instantaneous onset, (2) paralytic nature of the symptoms and (3) a tendency to disappear spontaneously. To these we would add: (4) a history of trauma and (5) the elimination of alcohol and drugs and other diseases.

Contusions of the brain were not mentioned by Vance. LeCount and Apfelbach[18] described them as "contre-coup bruises," and found them in 49.2 per cent of their cases. Jefferson defined the term contusion as "a general contusion of the whole brain," which is apparently what Vance, under the term concussion, called a "jarring of the brain";

14. Cassasa, Charles S. B.: Multiple Traumatic Cerebral Hemorrhages, Proc. New York Path. Soc. **24**:101, 1924.

15. Kaufmann, Edward: Pathology for Students and Practitioners, translated by Stanley P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 3, p. 1914.

16. Jefferson, Geoffrey: Discussion on the Diagnosis and Treatment of Acute Head Injuries, Proc. Roy. Soc. Med. **25**:742 (March) 1932.

17. Trotter, Wilfred: The Scalp, Skull and Brain, in Choyce, C. C., and Beattie, M.: A System of Surgery, New York, Paul B. Hoeber, Inc., 1923, vol. 3, p. 446.

18. LeCount, E. R., and Apfelbach, Carl W.: Pathologic Anatomy of Traumatic Fractures of the Cranial Bones and Concomitant Brain Injuries, J. A. M. A. **74**:501 (Feb. 21) 1920.

and Jefferson stated further that he implied "punctuate hemorrhages of greater or less extent." It seems that he includes the Cassasa type of hemorrhage also under this heading. Connors described contusion as "small hemorrhages in the pia arachnoid meshwork; and the patients have a bloody spinal fluid, but it is not as bloody as in laceration of the brain." Connors and McClure and Crawford [19] found contusion to be less frequent clinically than concussion, and this has been our experience. Under this heading we include all cases of subarachnoid or subdural hemorrhage without laceration of the brain. Subdural hemorrhage was present in 95 per cent of LeCount and Apfelbach's cases, and was most frequently due to lacerated cerebral veins.

Laceration of the brain has been the most common pathologic finding. It occurs with or without marked hemorrhage in all cases of severe trauma, and the spinal fluid is bloody. Lacerations may occur at the point of injury, or they may be contre-coup; contre-coup lacerations are the most frequent. The clinical symptoms in general in these cases are due to cerebral compression resulting from hemorrhage rather than from the laceration itself. Vance reported 132 deaths from cerebral compression and 24 deaths from laceration of the brain in his series. LeCount and Apfelbach did not use the term laceration, but spoke of such lesions as extensive bruises, and in a published photograph of a brain that exhibits a large laceration, they called the lesion a bruise. Apfelbach [20] in a later study found that the hemorrhage in contusion is greater the longer the patient lives, and thought that this is due to the persistence of the bleeding after the injury. This is true as regards lacerations of the brain, as shown by the fact that in some cases the spinal fluid may be clear immediately after an injury, whereas after a few hours it contains blood. We have yet to see a case of laceration of the brain in which the spinal fluid is persistently clear; in the vast majority of cases the fluid is bloody on lumbar tap immediately after the injury.

Extradural hemorrhage is due to a laceration of the middle meningeal artery or one of its branches in most instances, but Vance found rupture of the lateral sinus in two cases. He stated "that it occurs in thin skulls and the sharp edge of the bone lacerates the wall of the vessel." Epidural hemorrhage was present in 20 per cent of Vance's necropsies, and it was a death-causing factor in about 10 per cent of the necropsies which LeCount and Apfelbach reported, according to their opinion. In 1 of every 10 fatal cases epidural hemorrhage is the cause of death. A fracture is always present, and the vessel ruptures in almost all cases at the intersection of the path of the vessel and the

19. McClure, R. D., and Crawford, Albert S.: The Management of Craniocerebral Injuries, Arch. Surg. **16**:451 (Feb.) 1928.

20. Apfelbach, C. W.: Studies in Traumatic Fractures of the Cranial Bones, Arch. Surg. **4**:434 (March) 1922.

line of fracture. This is important when one operates on these patients. In only a single case in this series did the patient show the lucid interval usually described.

Cerebral edema is generally considered to follow immediately all severe cranial and intracranial injuries. The modern treatment of intracranial injuries is based on this concept. The explanation given is that the blood cells, freed from the lacerated vessels by the trauma, plug and also, by compression from without on the pacchionian granulations and arachnoid villi, prevent totally or partially the escape of cerebrospinal fluid into the venous sinuses, and the resultant back pressure on the brain of the spinal fluid produces increased intracranial tension and cerebral edema. It is a plausible explanation and may be a factor in some cases, but it is unsatisfactory in many ways; it fails to explain the cases in which there is definite hypotension of the spinal fluid, as described by LeRiche, and also cases seen at operation in which the brain is shrunken and not swollen. Cassasa denied the existence of cerebral edema due to trauma, because of the fact that the convolutions are not flattened like those seen in edema of the brain following infection or associated with tumors. LeCount and Apfelbach described a "traumatic edema," and in a later article Apfelbach stated "that it is a generalized edema, and not a localized one about contusions." Rand [21] found edema in his fatal cases, and later Rand and Courville stated that "there is an actual increase in the amount of circulating fluid." They also found edema in the choroid plexus in a number of fatal cases. The actual increase in the amount of circulating fluid can be due to subarachnoid hemorrhage. Jefferson found a characteristic histologic picture of edema in patients who lived until the second or third day, and he observed "dry" and "wet" brains after injury. In unilateral subdural hemorrhage Vance observed a flattening of the brain on the side opposite the hemorrhage, and in bilateral hemorrhages with laceration of the brain flattening of the cerebral cortex was not prominent unless one side showed a large quantity of blood while the other contained a small amount. Vance also found edema at times, after from two to five days, adjacent to the laceration, and he stated that "no case in this series presented the degree of swelling that Apfelbach mentioned, and there was no example of death arising solely from this condition." From studies of lumbar pressure we are of the opinion that the chief increase in intracranial tension may be explained on the basis of hemorrhage with resultant displacement of fluid. We do not deny entirely the existence of cerebral edema in cases of injury to the brain, but we do say, with Jefferson, that if it does occur it is a late development. And we feel, further, that it is not a chief, or even an important, consideration in the treatment of these patients. Alcohol as an edema-producing factor in these cases has not as yet been studied.

21. Rand, C. W., quoted by Rand and Courville.[12]

Unconsciousness or semiconsciousness was noted in 190 instances, or 54.4 per cent of our cases, as shown in table 4.

Unconsciousness was present in 61 of the 73 fatal cases, or 83.5 per cent, and 61 of the 190 unconscious patients died, or 32.1 per cent. Mental excitement was observed in many instances, and in some it was due to trauma plus alcohol. Traumatic delirium as described by Riddoch was also observed, but it was much less frequently noted than excitation. Alcoholism was the causative factor in some of the cases showing unconsciousness. In 12 fatal cases the patients were conscious on admission; death was caused in 5 by a terminal pneumonia, in 1 by meningitis, in 1 by epidural hemorrhage combined with laceration of the brain (this patient was operated on), in 1 by septicemia from a massive contusion of the back and in the remaining 4 by exhaustion. Most patients in the group with nonfatal fractures recovered from the unconsciousness within a few hours; in a few it lasted twenty-four hours, and in only 4 did it last as long as thirty-six hours.

TABLE 4.—*Incidence of Unconsciousness and Semiconsciousness*

	Class A		Class B			
	Craniocerebral Injuries		Group 1 Skull Fractures		Group 2 Intracranial Injuries	
	Lived	Died	Lived	Died	Lived	Died
Unconscious	24	19	17	22	19	6
Semiconscious	30	7	25	7	14	0
Conscious	45	4	56	6	44	2
Total	99	30	98	35	77	8

Cohen [22] in 1917 and Blakeslee [23] in 1929 reported on the findings in the eyes of patients with fractured skulls at Harlem Hospital. Blakeslee's report was especially comprehensive, and we studied many of the same patients on whom he made his observations. This series of cases was studied by us at a later time, and our findings confirmed his work. He reported a mortality of 95.5 per cent in patients showing bilateral widely dilated and fixed pupils, while it was 50 per cent in patients exhibiting a unilateral fixed pupil. Kearney [24] reported no change in the fundi during the first twelve hours after injury, but he reported changes in the fundi in 1 in 6 cases of severe injury after twelve hours. Papillitis occurred in our cases in few instances.

22. Cohen, Martin: The Value of Eye Manifestations Complicating Fractured Skulls, Arch. Ophth. **46**:258, 1917.

23. Blakeslee, George Arthur: Eye Manifestations in Fracture of the Skull, Arch. Ophth. **2**:566 (Nov.) 1929.

24. Kearney, J. A.: The Value of Eye Observations in Fracture of the Skull and Severe Head Injuries, New York State J. Med. **22**:341 (Aug.) 1922.

McCreery and Berry pointed out the value of using the pupillary signs as an indication of the patient's progress. As a rule, a unilateral dilated fixed pupil occurs on the same side as the lesion, but in one of our cases we observed a dilated fixed pupil on the side opposite to the laceration of the brain.

In a recent case of combined epidural hemorrhage and laceration of the brain, the pupil on the side of the laceration was dilated and fixed, while the pupil on the side of the epidural hemorrhage was smaller and reacted to light. Blakeslee was especially careful to point out the variation in the pupillary status as it occurred in different cases and as it changed from time to time in the same patient.

The incidence of the usual objective signs of injury to the head is shown in table 5.

The lacerations, contusions and hematomas of the scalp varied in number, extent and degree, and were valuable at times in furnishing a clue as to the nature of the trauma and the direction of the force in

TABLE 5.—*Incidence of Objective Signs of Injury*

	Class A	Class B		Total	
	Cranio-cerebral Injuries	Fractured Skulls	Intracranial Injuries	Number	Per Cent
Lacerations of scalp	26	55	24	105	30.2
Hematomas and contusions	22	28	27	77	22.1
Ecchymosis of eyelids	87	91	58	236	68.0
Bleeding from one or both ears	23	25	2	50	14.6
Bleeding from nose or mouth	27	15	17	59	14.3

relation to the head, which aided in localizing the lesion. Ecchymoses about the eyes were present in 68 per cent of these cases.

Bleeding from one or both ears is a rather reliable sign of fractured skull. Gurdjian [25] reported bleeding from the ears in 476 of 2,600 cases; his mortality in cases of unilateral bleeding from the ear was about 37.5 per cent, and in bilateral bleeding it was 67.3 per cent. Miller and Lauppe [26] reported mastoiditis as a sequel of fracture of the temporal bone in a case in which there was bleeding from the ear, and they found records of 22 similar cases; Gurdjian reported 3 cases in his series. Bleeding from the ear is not always due to a fracture of the skull, and this was true in 2 of our cases; in the first the bleeding was from a bleb on the wall of the external auditory canal, and in the second it was due to a simple rupture of the tympanic membrane. Vance cited a case in which the bleeding was from a laceration of the external auditory canal. The escape of spinal fluid from the ear makes

25. Gurdjian, E. S.: Ear Complications in Acute Craniocerebral Injuries, Radiology **18**:74 (Jan.) 1932.

26. Miller, R. K., and Lauppe, F. A.: Mastoiditis Following Skull Fracture, J. Michigan M. Soc. **29**:912 (Dec.) 1930.

the diagnosis of fracture of the skull certain, but in some instances the fluid is undetected owing to its admixture with blood.

The escape of cerebrospinal fluid from the ear was observed in 2 of our patients, both of whom died. Meningitis occasionally develops in cases of fracture involving the middle ear.

Bleeding from the nose and throat is of interest in that in unconscious patients some of the blood may flow into the trachea and bronchi and initiate pneumonia. It may also produce sounds in the trachea that simulate those of pulmonary edema. In 3 of our cases bleeding from the mouth was due to an associated fracture of the mandible.

The pulse rate varied so widely and was influenced by so many factors that we do not consider it a dependable guide. Many patients on admission showed a very rapid and feeble pulse owing to shock, and in many of the fatal cases the rapid pulse persisted until death. Bradycardia was of importance in some cases. It was present in 16 patients on admission; 13 of these patients died, while in the remaining 3 the pulse rate returned to normal within twenty-four hours. In 6

TABLE 6.—*Paralysis*

	Total Number of Cases	Nonfatal Cases	Fatal Cases
Cranial nerve paralysis	70	47	23
Facial nerve paralysis, alone	26	17	9
Hemiplegia	31	4	27
Monoplegia (exclusive of that of cranial nerve)	10	3	7
Motor aphasia	1	1	0

patients bradycardia developed after four days, persisted for several days and then disappeared. The changes in the volume of the pulse and the rapidity with which they occurred impressed us more than did the changes in the pulse rate.

The temperature on admission of the patients who were in a state of shock was subnormal. All patients who were not in a state of shock showed an average temperature range of between 100 and 101 F. The temperature showed wide variation within a few hours, and in the fatal cases, even in the absence of infection, it rose sharply to 105 or 106 F. just before death. In practically all of the nonfatal cases, irrespective of the temperature on admission, the temperature ranged between 99 and 101 F. for several days, and then descended to normal.

The neurologic aspects of these cases were studied by Dr. George A. Blakeslee and his associates. The incidence of various forms of paralysis is shown in table 6.

Dementia paralytica was observed in all fatal cases just before death. Abnormal reflexes were observed in most of these cases, and varied according to the site of the lesion. Convulsions were observed in 22 patients, 18 of whom died.

Vomiting is frequently mentioned as a symptom, but it was observed only 11 times in this series; in a single instance the vomiting was projectile.

Rigidity of the neck in the absence of meningitis was noted in 11 cases, and some of these cases exhibited a suggestion of a Kernig sign. Russell [27] observed rigidity of the neck 38 times in 200 cases, and stated that "it is often a sign of subarachnoid hemorrhage." Vance said: "The occurrence of a rigid neck and a positive Kernig sign can be caused by a subarachnoid hemorrhage." Ohler and Hurwitz [28] observed stiffness of the neck in 19 of 22 patients whose condition was diagnosed as spontaneous subarachnoid hemorrhage. They found the Kernig sign frequently present. We do not deny that subarachnoid hemorrhage may produce nuchal rigidity, but it seems to us that in some of our cases it could be explained by concomitant injury of the muscles of the neck at the time of the original injury and in others by the way the assistant bent the neck while the spinal puncture was being done, especially if the patient strained at this time, in some cases thereby producing muscle strain with or without a sprain of the ligamentum nuchae. This is supported by the fact that 2 of our patients showed a clear spinal fluid, and many patients with fracture of the occipital bone failed to show rigidity of the neck.

Urinary incontinence occurred frequently in this series, and we deemed it a most important prognostic sign. During the early part of this work, its occurrence was not always recorded, and so it is impossible for us to present complete figures. We have observed it in more than 50 cases. We feel safe in saying, however, that it occurred in the great majority of fatal cases, and that its occurrence in a patient not under the influence of alcohol is of grave prognostic significance. The beds of these patients are wet, and there is never incontinence of feces. If urinary incontinence persists in an alcoholic patient after the influence of alcohol has worn off, this persistence is in our opinion indicative of severe cerebral injury. The postmortem observations in cases showing urinary incontinence were laceration of the brain and compression of the brain from subdural or extradural hemorrhages.

Associated injuries were present in 23 instances, as outlined in table 7.

Readings of blood pressure were made on every patient as soon after admission as possible. The highest systolic pressure recorded was 240 mm. of mercury, with a diastolic pressure of 110 mm., while the

27. Russell, Ritchie: Discussion on the Diagnosis and Treatment of Acute Head Injuries, Proc. Roy. Soc. Med. **25**:751 (March) 1932.

28. Ohler, W. R., and Hurwitz, David: Spontaneous Subarachnoid Hemorrhage, J. A. M. A. **98**:1856 (May 28) 1932.

lowest pressure was so low that it could not be read (in this patient the reading was zero for about thirty minutes, after which it rose to 70 mm. systolic and 40 mm. diastolic, and it remained at or about that level until the patient died three days later). In 54 fatal cases the systolic pressure was above 130 mm. of mercury, and averaged about 150 mm., while in 11 fatal cases the systolic pressure was below 90 mm. In fatal cases the blood pressure readings were either above or below normal. No patient that recovered exhibited a systolic pressure below 90 mm., and most of the patients with nonfatal cases were found to have a blood pressure that was normal or nearly so. The relationships between the gradual increase in intracranial pressure and the blood pressure are definitely known, but we are not satisfied that the variations in blood pressure and its relationships in cases of cerebral trauma are as simple as is generally believed. We have occasionally found the

TABLE 7.—*Associated Injuries in Addition to Fractured Skulls and Intracranial Injuries*

	Number of Cases	Number of Fatal Cases
Fracture of:		
Femur	4	3
Mandible	3	1
Humerus	3	1
Ribs	4	3
Vertebrae	2	2
Pelvis	1	0
Tibia and fibula	2	2
Metacarpals	1	1
Clavicle	1	0
Patella	1	0
Ruptured spleen	1	1
Massive contusion of back	1	1
Total	24	15 or 62.5%

same mechanism at work in these cases of trauma as in cases of tumor of the brain, but we feel in general that there are one or more added factors due to the trauma and also that there is insufficient time for the various compensatory and regulatory mechanisms of the cerebrospinal and circulatory systems to get into play, as they undoubtedly do in cases of slow cerebral compression; therefore the process is extremely complicated and difficult to interpret, except in its obvious phases.

Diagnostic spinal taps are made as a routine on all patients immediately after their admission to the hospital. This procedure is simple and harmless when properly done, and we justify its routine use, after admitting that in an occasional case harm may result, on the grounds that in many cases acute craniocerebral injury would otherwise be undiagnosed and untreated. The Ayer water manometer is used for all pressure readings. We discarded the small mercury manometer after using it for several years, as it was not as accurate or reliable as the water manometer. Our findings are shown in table 8.

Jackson,[29] Sharpe,[30] Rodman,[31] Fay [32] and many other serious workers have stressed the value of determinations of spinal fluid pressure as a guide to treatment. In the average fatal case the intraspinal pressure was above normal; on the other hand, not infrequently we found hypotension. In a few cases readings were not made because of the death of the patient soon after admission to the hospital, and a few patients refused to have the determination made. It is unwise to place too much emphasis on lumbar pressure readings, owing to the fact that the rise and fall of intraspinal pressure often occur suddenly and without apparent cause; and, too, very slight changes produce marked variations in the readings, as for example, respiration and slight changes in position. Many cases in which injury was suspected and which later proved to be instances of alcoholism showed high pressure although there was no injury. In the absence of other obvious clinical signs, these readings have aided us little as regards prognosis.

TABLE 8.—*Spinal Fluid Pressure Readings*

Spinal Fluid Pressure Readings in Mm. of Water on Admission to Hospital	Class A		Class B			
	Craniocerebral Injuries		Group 1 Skull Fractures		Group 2 Intracranial Injuries	
	Lived	Died	Lived	Died	Lived	Died
0 to 50	1	3	10	4	4	2
50 to 120	57	6	32	2	31	2
120 to 200	23	9	38	6	15	2
200 to 300	11	3	7	5	14	1
300 to 500	2	2	4	5	4	1
500 to 700	0	3	1	5	1	0
Total	94	26	92	27	69	8

After the spinal fluid pressure reading is made, about 5 cc. of spinal fluid is collected separately in three test tubes, and the fluid in the third test tube is sent to the laboratory for a red and a white cell count. The red cell count is recorded on every patient's chart. We no longer speak of a "pink" or a "bloody" spinal fluid, but describe it in terms of the red cell count. This is done in the interest of greater accuracy. An opalescent spinal fluid was observed 8 times. Erythrocyte and leukocyte counts of the spinal fluid were made in 85 nonfatal and in 47 fatal cases of proved fractured skull. In the 85 nonfatal cases the average red cell count was 39,289, while the average white cell count

29. Jackson, Harry: The Management of Acute Cranial Injuries by the Early, Exact Determination of Intracranial Pressure and Its Relief by Lumbar Drainage, Surg., Gynec. & Obst. **34**:484 (April) 1922.

30. Sharpe, William: Diagnosis and Treatment of Brain Injuries with and without Fracture of the Skull, Philadelphia, J. B. Lippincott Company, 1920.

31. Rodman, J. Stewart: Editorial, Surg., Gynec. & Obst. **48**:437 (March) 1929; Surgical Management of Cranial Injuries, Ann. Surg. **92**:1017 (May) 1931.

32. Fay, Temple: Head Injuries. The Results Obtained with Dehydration in Forty-Eight Consecutive Cases, J. Iowa M. Soc. **20**:447 (Oct.) 1930.

was 653, and the average differential count was polymorphonuclear leukocytes 60 per cent and lymphocytes 37 per cent. The hemoglobin, according to the Sahli method, averaged 13 per cent. In the 47 fatal cases the red cell count averaged 857,813, the white cell count averaged 1,170, and the differential count averaged polymorphonuclear leukocytes 65 per cent and lymphocytes 34 per cent; the average hemoglobin was 21 per cent. It is seen that the more serious the injury, the higher was the red cell count, as well as the white cell count and the percentage of hemoglobin.

Urinalyses were made on 318 patients, and in many cases several examinations of the urine were made. Dextrose was found in the urine of 19 patients, and of these 19 patients 6 died, or 31.5 per cent. All patients that were conscious were allowed to void urine, while unconscious patients were catheterized. In cases of incontinence a urinal was adjusted to the penis to collect the urine. Determinations of blood sugar were made on 35 unconscious patients; the amount was above 120 mg. in 12 patients, or 31.4 per cent. Seven of the 12 cases exhibit-

TABLE 9.—*Erythocyte and Leukocyte Counts in Cases of Fractured Skull*

	142 Nonfatal Cases	54 Fatal Cases
Red cell count, average	4,464,436	4,398,704
White cell count, average	9,548	12,957
Differential count, average		
Polymorphonuclears	79%	62%
Lymphocytes	24%	21%
Hemoglobin	81%	77%

ing hyperglycemia were fatal. The blood sugar level was normal or below normal in the remaining 23 cases. The blood for the determinations of blood sugar was taken immediately after the arrival of the patient in the ward. Our findings are, therefore, at variance with those of Mock and de Takats,[33] who stated that "head injuries of sufficient severity to cause unconsciousness result constantly in hyperglycemia." Their opinion is based on an experimental study and observations made in 10 clinical cases. Only 3 of the cases that showed hyperglycemia showed sugar in the urine. We made subsequent determinations of blood sugar at twenty-four hour intervals on the 12 hyperglycemic patients, and in only 2 instances did the blood sugar level remain above normal for over twenty-four hours.

Erythrocyte and leukocyte counts were made in 142 nonfatal cases of proved fracture of the skull and in 54 fatal cases of fractured skull. The average counts are shown in table 9.

The blood was examined within four hours after the patient's admission. Cases showing associated injuries were excluded from

33. Mock, Harry E., and de Takats, G.: Hyperglycemia Following Head Injuries, Ann. Surg. **90**:190 (Aug.) 1929.

this group. There was a higher white cell count in the fatal cases, but our figures are not nearly as high as those obtained by Wright and Livingston.[34] Wright and Livingston have called attention to the diagnostic value of leukocytosis in intradural hemorrhage, and Moody [35] considered it of diagnostic value in extradural hemorrhage.

Roentgen examination is one of the most important examinations to be made in these cases. Our routine is to have roentgenograms made while the patient is in transit from the office of admission to the wards, unless he is in a state of very deep shock. Unconscious patients must be handled with extreme gentleness, and exposures made with rapidity by one skilled in taking roentgenograms of the skull. The diagnosis of fracture of the skull can be made in living persons with certainty only by means of roentgen examination, except in cases of penetrating wounds of the skull, which are usually compounded and depressed and in which one can see directly that the skull is fractured. Stereoscopic plates should be made in each case, and we advise the taking of views of the frontal and occipital and of the two lateral regions of the skull, making eight films in all. We do not try to get a roentgenogram of the base of the skull, because in our opinion it is dangerous. In our experience most basal fractures extend upward onto the lateral walls for a sufficient distance for them to be detected in an ordinary eight film study. Flat plate studies are practically worthless, as they do not give a proper concept of the fracture if one is found, and, too, it is impossible in many instances to differentiate suture lines and vessel markings from fractures. In fractures of the frontal sinus it is impossible to tell on a flat plate which wall of the sinus is fractured. We obtained roentgenograms showing evidence of fracture in 263 of the 347 cases, or 75 per cent. Stewart [36] reported a roentgenologic study of 300 cases of so-called "head injuries" from Harlem Hospital in 1925, in which 20 per cent of the cases showed evidence of fracture. Fay reported an incidence of 24.8 per cent of fractures proved by roentgen examination and seen at operation in a series of 189 cases.

In only 2 of our 35 fatal cases, in all of which autopsy was performed, did the autopsy disclose a fracture that we were unable to find on roentgen examination. We were able to make the diagnosis of combined epidural and subdural hemorrhage in 4 cases by means of roentgenologic study (the diagnosis of epidural hemorrhage was based on the fact that there was comminution of bone over the groove of the

34. Wright, Arthur M., and Livingston, Edward M.: The Leucocytosis of Internal Hemorrhage, New York State J. Med. **23**:286 (July) 1932.

35. Moody, W. B.: Fracture of Cranial Bones, with Especial Reference to Extradural Hemorrhage, J. A. M. A. **74**:511 (Feb. 26) 1920.

36. Stewart, William H.: Skull Fractures, New York, Paul B. Hoeber, Inc., 1925, p. 5.

middle meningeal artery). Three of these patients were operated on; 2 died and 1 lived. Autopsy in the 2 fatal cases in which operation had been performed and in the fatal case in which there had been no operation showed a large laceration of the brain complicating the extradural hemorrhage. Luckett [37] stated several years ago that symptomless fractures are relatively common after even apparently trivial violence, and urged the routine use of roentgen examination in all instances in which injury of this type is suspected. Encephalograms have been made in some of these cases, and we can testify to their value in giving information regarding permanent damage done by the trauma, as was first pointed out and demonstrated by Pancoast and Fay.[38] One of Grant's [39] cases of chronic subdural hematoma showed the value of roentgen examination in helping to make the diagnosis of this condition at times; we did not have any case of this kind in this series, however.

In line with the pioneer work of Connors, we used conservative nonoperative treatment. In the cases in which operation was not performed it consisted chiefly of rest in bed, good nursing and the maintenance of nutrition. Early in our work we used intravenous injections of hypertonic solution of dextrose in 20 seriously injured patients, 19 of whom died; we stopped using this treatment, as we are now of the opinion that the patients who live after receiving injections of dextrose would live without them, and in some instances it seemed to us that the injections hastened death. Death in these cases is due to cerebral compression from hemorrhage caused by laceration of the brain, and as dextrose cannot stop hemorrhage or heal a lacerated brain its use seems illogical.

Browder's [40] case demonstrates this point, and Jefferson has had the same experience in some of his cases. We agree that hypertonic solution of dextrose will shrink the brain, but this is a temporary effect, and the shrinkage that occurs allows space for more bleeding and a subsequently larger compressing mass. Recent experimental work on dogs by Milles and Hurwitz [41] showed that the reduction of intracranial pressure by single doses of hypertonic solutions of salt and dextrose

37. Luckett, William H., in Stewart, William H.: Skull Fractures, New York, Paul B. Hoeber, Inc., 1925, p. 15.

38. Pancoast, H. K., and Fay, Temple: Encephalography: Roentgenological and Clinical Considerations for Its Use, Am. J. Roentgenol. **21**:421 (May) 1929; Encephalography as the Roentgenologist Should Understand It, Radiology **15**:173 (Aug.) 1930.

39. Grant, Francis C.: Chronic Subdural Hematoma, Ann. Surg. **86**:485 (Oct.) 1927.

40. Browder, Jefferson: Dangers in the Use of Hypertonic Solutions in the Treatment of Brain Injuries, Am. J. Surg. **8**:1213 (June) 1930.

41. Milles, George, and Hurwitz, Paul: The Effect of Hypertonic Solutions on Cerebrospinal Fluid Pressure, with Special Reference to Secondary Rise and Toxicity, Arch. Surg. **24**:591 (April) 1932.

is transient, and that a secondary rise in pressure follows which may occasion death. Weed and his co-workers'[42] original work was done on cats with normal brains, and not brains that had been subjected to severe trauma and compressed by blood clots. We can testify to the fact that in the human subject hypertonic dextrose solutions will produce an almost instantaneous shrinkage of the brain. This was seen during an operation on a patient who showed focal signs of pressure; when we opened the dura, the arachnoid seemed full of fluid and the brain bulged into the opening (at this time 50 cc. of a 50 per cent solution of dextrose was given intravenously, and immediately the brain shrank and its convex surface became concave, as if the ventricle on that side had suddenly emptied itself of fluid). Solutions of magnesium sulphate were not used in the rectum, because previous experience at this hospital had shown this treatment to be unsatisfactory.

Lumbar drainage was used in only 15 cases, and we do not advocate its use. In November, 1931, we used lumbar drainage combined with

TABLE 10.—*Operations*

Total number of cases	347
Total number of operations	18
Compound depressed fracture (1 death or 12.5%)	8
Simple depressed fracture (no deaths)	4
Combined epidural and subdural hemorrhage, dura not opened (3 deaths or 75%)	4
Subdural hemorrhage with focal signs (2 deaths or 100%)	4
Total number of deaths (33.5% mortality)	6

limitation of the intake of fluid, as suggested by Fay, in 15 patients, and these patients seemed so much worse clinically than is usual that we discontinued the use of drainage and stopped limiting the intake of fluid. The idea has grown with us, since we have heard of so many permanent poor end-results in other clinics, that the drying of the brain and spinal cord and of the leptomeninges may, since it is unphysiologic, contribute no small amount to such an unsatisfactory end-result. We are trying to check as many of our end-results as possible by means of encephalograms. We have used phenobarbital and amytal with good results in allaying excitement due to cortical irritation. In 2 cases we

42. Weed, Lewis H., and McKibben, Paul S.: Pressure Changes in the Cerebrospinal Fluid Following Injection of Solutions of Various Concentrations, Am. J. Physiol. **43**:512, 1919. Weed, Lewis H., and Hughson, Walter: Systemic Effects of the Intravenous Injection of Solutions of Various Concentrations with Reference to the Cerebrospinal Fluid, ibid. **58**:53, 1921; The Cerebrospinal Fluid in Relation to the Bony Encasement of the Central Nervous System as a Rigid Container, ibid. **58**:85, 1921; Intracranial Venous Pressure and Cerebrospinal Fluid Pressure as Affected by Intravenous Injection of Various Concentrations, ibid. **58**:101, 1921.

have used avertin with good results. Patients in a state of shock are given fluids by mouth and by hypodermoclysis, the amount varying with each case.

The patient is kept warm at all times, and suction is used to keep the throat clear of blood and mucus. In unconscious patients nutrition is maintained by means of duodenal feedings and nutritive enemas. The bed is put in Fowler's position, and elderly patients are placed on an air mattress.

Operations were performed in all cases of compound depressed fractures, in some of simple depressed fractures, and in those with epidural or suspected epidural hemorrhage. No patients with fractures of the frontal sinus were operated on, although when we had a case in which we were certain that the dura was torn we operated and closed the dura, as suggested by Teachenor.[43] No operation was performed for cerebral compression, as it is our belief that if the patient lives he would have lived without operation. Eighteen operations were performed in this series, as shown in table 10.

CONCLUSIONS

1. This study confirms the work of others showing that the nonoperative treatment of fractured skulls is the best treatment known today.

2. Much more intensive investigation is needed to lower still further the mortality in these cases.

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43. Teachenor, F. R.: Intracranial Complications of Fracture of the Skull Involving Frontal Sinus, J. A. M. A. **88**:987 (March 26) 1927.

FIBROMYOMA OF THE UTERUS

REPORT OF A CASE OF A SIXTY-FIVE POUND SOLID FIBROMYOMA, WITH A REVIEW OF LARGE CYSTIC AND SOLID UTERINE MYOMAS

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My object in writing this paper is to place on record the operative removal of an exceptionally large myoma of the uterus, and at the same time to learn, if possible, from a review of similar cases, if anything further could have been done to promote a more successful result. I was also interested to see how this tumor compared in size with others reported in the literature and what the mortality in a series of such cases would be. Knowledge of mortality is very important, because the question always arises in the mind of both the patient and the surgeon whether it is worth while to attempt operation when the tumor has reached such huge proportions. I felt that I might pick up some details of technic, at operation, in preoperative or postoperative care, that might help me should a similar case present itself. Therefore, the literature on this subject was reviewed, as far as possible, back to the beginning of the period of aseptic surgery. I set 30 pounds (13.6 Kg.) as the arbitrary minimum weight limit of the tumors to be included in this series, and was able to collect over thirty cases that had complete enough records to make them valuable.

REPORT OF A CASE

History.—Mrs. Dora C., aged 56, a housewife, was admitted to the service of Dr. T. S. Cullen at the Church Home and Infirmary, Baltimore, on Jan. 31, 1931. Her chief complaint was swelling of the abdomen of twenty years' duration. The family history was essentially unimportant. Her menstrual periods began at the age of 13, lasted from three to four days, were never painful or excessive in amount, and ceased completely nine years before the present admission. Since this time there had been no discharge of any sort and no pelvic pain. The patient had been very dyspneic on even the slightest exertion for as long as eight years and had been gradually getting worse in this respect as the tumor had enlarged. She had had to sleep on several pillows for some time, and lately had had to sit up at night. She had been constipated, but otherwise the gastro-intestinal system had been normal. There had been no urinary symptoms of any sort.

The present illness began twenty-one years ago, following an abortion; the patient's family doctor noticed that there was a small hard lump in the right lower quadrant of the abdomen. He felt that this was probably a tumor of the uterus and advised operation, but she refused to have anything done. The mass continued to grow, moving upward and outward. No pain or other discomfort was associ-

ated with its increase in size, until lately. As the mass increased in size and tension became greater on the abdominal wall, a small ventral hernia which had been present increased gradually to a very great size until it reached nearly to the patient's knees, hanging down like an inverted cone with the base uppermost. Four years ago, because of the weight and appearance of the tumor, the patient sought the advice of Dr. Cullen. He advised surgical removal, but she again refused operation. For the past two years she had been a semi-invalid because

Fig. 1.—Front view of Mrs. Dora C. to show position of the lobules of the tumor preoperatively.

of the weight of the tumor and shortness of breath. Three weeks ago she had a period when she suffered marked chills and fever, and the abdominal tissues over the most dependent portion of the tumor became very red and sore. Hospitalization was advised, and after much hesitancy the patient finally consented to be admitted.

Physical Examination.—The patient was well developed and nourished and was lying in bed on her right side, propped up with pillows to an angle of about 45 degrees. Her color was not very good, and respirations were rapid and rather

shallow. The general physical examination gave essentially negative results except for the cardiorespiratory system. There were slight enlargement of the heart, a few moist râles at the bases of both lungs and edema of the lower portion of the abdomen, legs and ankles. However, the respiratory rate was only 24 and the pulse rate, 90. The blood pressure was 100 systolic and 65 diastolic.

The abdomen presented the chief point of interest. There was a huge umbilical hernia which extended in a conical form 11 inches (27.9 cm.) below the symphysis pubis and was from 6 to 9 inches (15 to 22.8 cm.) in diameter. The walls of the abdomen and the hernia were very tense and showed dependent edema. The abdomen measured at the waist 55 inches (139.7 cm.), whereas normally the patient said her waist measured only from 32 to 34 inches (81 to 86 cm.). The abdomen was so tense and there was so much edema present that no good idea of the contents could be gained at this time. A little clear fluid was leaking from a point where a paracentesis had been made. Rectal examination gave negative

Fig. 2.—Lateral view of Mrs. Dora C. to show position of the lobules of the tumor preoperatively.

results. Pelvic examination showed relaxation of the outlet. Through the vagina no tumor mass could be made out because of the fluid present. It looked very much as if a large ovarian cyst with ascites was being dealt with.

On February 2, after more fluid had been drawn off and more leakage had occurred, a huge, irregular, hard mass could be felt filling the entire abdomen and also the large hernial sac. The tumor was clearly an unusually large fibroid and not an ovarian cyst.

After several days, during which time the patient was digitalized and hot compresses were applied to the area of induration of the abdominal wall until it had completely cleared up, operation was decided on, as the maximum preoperative improvement had been reached. The patient was clearly not a good risk, but something had to be done if any improvement could possibly be expected.

Operation (Dr. Cullen, February 7).—An elliptic incision was made, extending from one side to the other and embracing the hernia. The breadth of the ellipse was about 18 cm. The flap was gradually dissected free and the hernial ring exposed. It was 14 cm. across. The hernial sac contained a large myoma. When

the sac was opened still further, a second myomatous nodule appeared, then a third, and then a loop of large bowel which was adherent to the sac. Omental vessels ran into the surface of the tumor.

After the hernia was reduced and its contents freed, the large abdominal tumor was gradually delivered. There were five lobes in all. After the tumor was delivered, the vessels were tied from left to right; the left tube and ovary were not removed. It was difficult to get one's bearings. Down the right side was a lobule 12 cm. in diameter. It was situated in the broad ligament and was exceedingly difficult to enucleate. When the vessels were being clamped, a white cord was seen which appeared to be the right ureter and which finally proved to be this structure. How it was avoided, I do not know. The entire mass was removed, and all bleeding points were checked, but there was troublesome oozing in the broad ligament on the right side where the myoma had been shelled out. Two cigaret drains were left in this ligament and brought out through a right gridiron incision.

The appendix was tied up in adhesions and thickened; it was removed. The abdomen was then closed.

The patient stood the operation quite well until the last fifteen or twenty minutes, when her pulse became rapid and very weak. She received nearly 500 cc. of salt solution intravenously on the table and an infusion was also started. When she left the table her pulse was barely perceptible and could not be counted. Her color was poor, and she felt cold and clammy. The wound was dressed in the usual manner and a tight abdominal binder applied. No fluid was put into the abdomen.

Postoperative Course.—Following the operation the patient was temporarily moved into a side room still on the operating table, the head lowered, the body surrounded by hot water bottles and wrapped in warm blankets, and a transfusion of 500 cc. of citrated blood was given. During the succeeding twenty-four hours she received 1,000 cc. of coffee and saline by rectum in divided doses of 250 cc. each. Physiologic solution of sodium chloride by infusion and intravenously, 2,050 cc., 1 grain (0.065 Gm.) of morphine, ¾ grain (0.048 Gm.) of ephedrine (hypodermically), 16 minims (1 cc.) of epinephrine (hypodermically), 58 grains (3.75 Gm.) of caffeine in doses of 7½ grains (0.49 Gm.) and 2 ampules of camphor in oil. At the end of this time she had failed to rally. Her pulse was 140, respirations, 24, and temperature, 101.6 F. On the following day, February 8, this same treatment was repeated together with another transfusion of 500 cc. of blood, but still the condition of shock persisted, and although the patient was conscious and talking rationally during most of this period, her condition gradually became worse, so that by midnight the temperature was 102.6 F.; the pulse rate, 140, and respirations, 44; she finally died at 2:35 a. m., February 9. It seems remarkable that in spite of all our efforts we were never able to overcome the condition of shock which set in during the last few minutes of operation, although the patient lived nearly forty-eight hours. Permission for complete autopsy was obtained, but did not show anything more than we already knew, as all the organs seemed entirely normal except for slight enlargement of the heart and a little edema at the bases of the lungs.

Gross Description of the Tumor (Dr. Norwood).—The specimen was that of a huge lobulated fibromyoma of uterine origin. It measured 30 by 60 cm. and weighed 65 pounds (29.5 Kg.). There were no areas of degeneration on the cut surface. No structure could be definitely identified as being uterine or ovarian tissue. One flattened out structure on the anterior surface of the tumor looked like a tube.

Microscopic Examination.—Examination under the microscope showed perfectly uniform typical fibromyomatous tissue with no degenerative changes.

A comprehensive analysis of the foregoing cases is not possible because of the many factors involved, but when one looks over the list superficially certain things stand out. Evidence of severe involvement of the cardiovascular system of the patients preoperatively, such as dyspnea, orthopnea, enlargement of the heart and edema at the bases of the lung, has usually been an indication that the patient would not

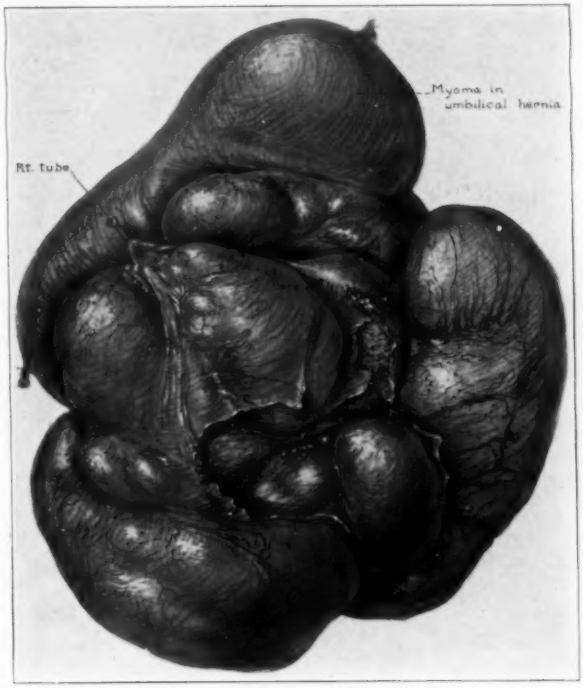

Fig. 3.—Specimen immediately after operation.

survive operation. The time consumed at operation and the fact as to whether the release of abdominal pressure was counteracted by external means or by the use of intra-abdominal fluid, such as physiologic solution of sodium chloride, have also been two indicators of the outcome, for in those cases in which operation was performed quickly and in which fluid was used in preference to external pressure, the mortality rate has been lower. The consumption of less time and the use of intra-abdominal fluid might have given our patient a better chance, whereas the fact that she had cardiac enlargement, edema at the bases of the lungs and severe dyspnea with rapid pulse on the slightest exertion

Author and Date	Age of Patient	Duration of Symptoms	Condition of Patient Before Operation	Duration of Operation	Weight and Character of Tumor	Fluids Given at Operation	Result
Wells, T. S.: Brit. M. J. **1**: 674, 1878..	36	10 yrs.	Good except for edema of legs and feet during menses	1 hour	68 lbs., 6 oz.; solid fibromyoma	None	Excellent recovery
Tait: Diseases of Women and Abdominal Surgery, ed. 2, New York, William Wood & Company, 1879, vol. 1, p. 187	63	26 yrs. (?)	Dyspnea; weight interfered with work as midwife	?	26½ lbs.;* solid fibromyoma	.....	Uneventful recovery
Dudley, E. C.: New York M. J. **39**: 697, 1884	47	5 yrs.	Poor, cardiac weakness; emaciation; anemia	?	35 lbs.; solid fibroid	None	Hectic course; recovery
Stockard, C. C.: M. Rec. **26**: 177, 1884	54	12 yrs.	Poor, emaciation; cardiac distress	No operation	135 lbs. (?)†; cystic fibroid	.....	Died 6 days after tapping
Thornton, K.: Lancet **1**: 672, 1887....	56	5 yrs.+	General condition fair; details not given	?	30½ lbs. (approx. 5 lbs. fluid); solid fibroid	None	Slow but complete recovery
Bantock: M. Press & Circ. **46**: 645, 1888	..	10 yrs.	Cardiac distress and albuminuria	Not given	64 lbs.; solid fibroid	.....	Died 6 days after operation; kidney failure (?)
Hunt, S. H.: Am. J. Obst. **21**: 62 1888	53	21 yrs.	.....	Removed at autopsy	140 lbs.; fibroid with many cysts		
Werder, X. O.: New Orleans M. & S. J. **17**: 247, 1889-1890	23	8 mos. (?)	Good	?	40 lbs. (estimated); fibrocystic	None	Good recovery
McIntyre: St. Louis M. & S. J. **60**: 137, 1891	38	?	Respiratory and cardiac distress	?	93½ lbs.; solid fibromyoma	.....	Died 5 days after operation; septicemia
Doran, A.: Med.-Chir. Tr., London, **76**: 325, 1892-1893	..	.....	.....	?	42⅛ lbs.; fibrocystic	.....	Good recovery
Eastman, J.: North American Pract. **5**: 387, 1893	..	.....	.....	?	60 lbs.; solid fibroid	.....	Recovery (?)
Dalziel: Glasgow M. J. **41**: 229, 1894...	38	1 yr. (?)	Good	?	33 lbs.; cystic myoma; 13 lbs. fluid	.....	Good recovery
Penrose, C. B.: Am. J. Obst. **35**: 106, 1897	39	16 yrs.	Cardiac and respiratory difficulty for 4 years	45 minutes	87 lbs.; solid fibroid (edematous)	.....	Died from shock 4 hours after operation
Ricketts: Cincinnati Lancet-Clin. **41**: 531, 1898	52	4 yrs. (?)	Not given	58 minutes	65 lbs.; solid myoma	None	Result not given
Gwinard: Bull. et mém. Soc. de chirurgiens de Paris **28**: 201, 1902	40	23 yrs.	Good, except bedridden by weight	45 minutes	72⅝ lbs.; solid myoma	.....	Died of shock during night
Madden, F. C.: Brit. M. J. **1**: 70, 1902	28	2 yrs.+	Good	?	53 lbs.; soft, fleshy myoma	Filled abdomen with saline solution before closure	Uneventful recovery
	41	10 yrs.	Some dyspnea, other-	2.5 hours	87 lbs.; fibrocystic	Filled abdomen with	Uneventful

Webster, J. C.: J. Obst. & Gynæc. Brit. Emp. **4**: 135, 1903	41	10 yrs.	Some dyspnea, otherwise good; 55 per cent hemoglobin	2.5 hours	87 lbs.; fibrocystic	Filled abdomen with saline solution before closure	Uneventful recovery
Brown: Lancet **1**: 868, 1904.....	54	22 yrs.	Good until 3 weeks before death	None	70 lbs.; solid fibroid	.....	Tumor removed post mortem
Souligoux: Bull. et mém. Soc. de chirurgiens de Paris **33**: 1058, 1907	..	.....	.....	?	74 lbs.; cystic, 28 liters of fluid	.....	Recovery
Cullen, T. S.: J. A. M. A. **48**: 1491 (May 4) 1907	58	20 yrs.	Edema of abdomen and legs; could not lie down because of weight of tumor	55 minutes	89 lbs.; cystic myoma	No note as to fluid	Good recovery; lived nearly 20 years
Packard, H.: New England M. Gaz. **43**: 215, 1908	..	.....	.....	?	44 lbs.; solid myoma	.....	Recovery
Cullen, T. S., in Kelly and Cullen: Myomata of the Uterus, Philadelphia, W. B. Saunders Company, 1909, p. 118	48	4 yrs.	Good	?	39 lbs.; multicystic myoma resembling multi-ocular ovarian cyst	Some free fluid	Recovery
Cocram, H. S.: New Orleans M. & S. J. **43**: 591, 1910-1911	48	8 yrs. (?)	Bedridden by weight, but otherwise good	?	95 lbs.; solid myoma	.....	Uneventful recovery
Pakouski: Bull. et mém. Soc. anat. de Paris **87**: 375, 1912	..	.....	Good	.....	30 lbs.; solid myoma	.....	Uneventful recovery
Barry, C. C.: Indian M. Gaz. **48**: 310, 1913	44	18 yrs.	Good except for dyspnea	?	40 lbs.; myoma with small cysts	1,000 cc. soline sol. while on table; none in abdomen	Uneventful recovery
Jones, E. L.: U. S. Nav. M. Bull. **15**: 558, 1921	65	20 yrs.	Good	?	31 lbs.; cystic tumor (may be ovarian, no microscopic report)	.....	Uneventful recovery
Farmer, C.: Kentucky M. J. **21**: 543, 1923	..	.....	.....	?	30 lbs.; fibromyoma	.....	Uneventful recovery
Parkinson, W. R.: Brit. M. J. **2**: 1055, 1924	30	8 yrs.	Good	?	33 lbs.; soft fibromyoma	None	Good recovery
Stevens, T. G.: J. Obst. & Gynæc. Brit. Emp. **32**: 729, 1925	59	20 yrs.	Dyspnea; edema of legs	35 minutes	47 lbs.; solid myoma	None, but tight abdominal binder used	Good recovery
Hicks, E. S.: Canad. M. A. J. **18**: 58, 1928	..	.....	Good	1 hour +	65 lbs.; solid myoma	3 gal. of sterile water in abdominal cavity before closure	Good recovery; left hospital in 26 days
Gerich, C. O.: Zentralbl. f. Gynäk. **53**: 1652, 1929	48	20 yrs.	Good	?	33 lbs.; solid myoma	.....	Good recovery
Behrend, M.: Am. J. Obst. & Gynec. **20**: 699, 1930	35	.....	Good, except bedridden because of weight	?	133 lbs.; myoma with many small cysts	300 cc. of citrated blood on table	Died of pneumonia in 48 hours (?)

* The tumor weighed 26½ pounds (12 Kg.) after standing twenty-four hours, yet the author calculated it to have weighed 68 pounds (30.8 Kg.) at time of removal, which does not seem reasonable.

† The author removed 71 pounds (32.2 Kg.) of fluid from the cyst by tapping six days previous to the death of the patient, but he added this to the postmortem weight for the total of 135 pounds (61.2 Kg.). For this reason the tumor probably weighed considerably less than he calculated.

would suggest that following the general rule the chances for a favorable outcome were poor from the beginning.

The mortality rate of the entire series is 21.4 per cent when the tumors removed post mortem and those cases in which the outcome was uncertain are excluded. To me this seems surprisingly low, considering the size of the tumors, the age of the patients and their general condition. This low mortality rate certainly indicates that the surgeon is entirely justified in urging operation on patients of this type, for the outcome if operation is not performed is certain death, whereas operation promises a favorable result in a high percentage of cases.

When the tumor in the case reported here is compared to the others of the same type (solid fibromyoma), it is found that six others have been reported that were larger (Wells, 1878, 68½ pounds [31 Kg.]; McIntyre, 1891, 93½ pounds [42.4 Kg.]; Penrose, 1897, 87 pounds [39.5 Kg.]; Gwinard, 1902, 72⅗ pounds [33 Kg.]; Brown, 1904, 70 pounds [31.8 Kg.]; Cocram, 1910, 95 pounds [43.1 Kg.]. In these cases three of the patients made an uneventful recovery and three died. Two more cases are reported in which the tumors were of exactly the same weight (Ricketts, 1898, and Hicks, 1928), in both of which the patients made an uneventful recovery. This review did not show me anything more than that which is common knowledge in regard to the preoperative and postoperative care in these cases.

DOUBLE GALLBLADDER

REPORT OF A CASE

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AND

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Before the advent of cholecystography, clinical recognition of anomalies of the gallbladder was rarely recorded. Boyden [1] reviewed the subject of accessory gallbladder in 1926. His figures are based on the study of 10,000 domestic animals and 19,000 human beings. He found that this anomaly occurred once in every 8 cats, 28 calves, 85 sheep and 198 pigs and only once in from 3,000 to 4,000 human beings. Only 20 cases in human beings were recorded from 1674 to 1926. Boyden classified these into (1) vesica divisa, or bilateral gallbladder, and (2) vesica duplex, which was subdivided into: (*a*) Y-shaped, with two cystic ducts merging into one, and (*b*) ductular, with two complete cystic ducts opening separately into the common duct.

There were only 4 examples of the ductular variety in his series of 20 cases. Since the time of Boyden's publication, several more cases of this anomaly have been recorded. Priesel,[2] in 1927, reported a case and reviewed the older literature. Other cases of the ductular variety have been reported by Garofalo,[3] Braun,[4] Wolfson [5] and Holderman.[6] Wischnewsky [7] reported a case of the vesica divisa variety and Wakeley [8] a case in which there was apparently a Y-shaped bladder with two cystic ducts merging into one.

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1. Boyden, E. A.: Accessory Gall-Bladder, Am. J. Anat. **38**:177, 1926.

2. Priesel, A.: Verdoppelung der Gallenblase beim Menschen, Virchows Arch. f. path. Anat. **265**:76, 1927.

3. Garofalo, F.: A Case of Double Gall-Bladder, Bull. d. sc. med. Bologna **5**:380, 1927.

4. Braun, A.: Doppelbildung der Gallenblase, Zentralbl. f. Chir. **53**:1055, 1926.

5. Wolfson, W. L.: Supernumerary Gall-Bladder, Am. J. Surg. **6**:88, 1929.

6. Holderman, H. H.: Double Gall-Bladder, Ann. Surg. **91**:475, 1930.

7. Wischnewsky, A. W.: Doppelgallenblase während der Operationaufgedeckt, Arch. f. klin. Chir. **135**:779, 1925.

8. Wakeley, C. P. G.: A Double Gall-Bladder Removed by Operation, Brit. J. Surg. **15**:334, 1927.

Besides these anatomically described cases, 5 cases of double gallbladder have been reported by Nichols,[9] Climan,[10] Hayes [11] and Cave,[12] in which the diagnosis was made by cholecystography. These cases were apparently of the ductular variety, but only in Nichols' case was the organ removed surgically. The undoubted number of such cases since Boyden's summary, therefore, is 6 or, including his 4 cases, a total of 10. The other 4 cases discovered by means of cholecystography may or may not belong in this classification. We are adding another case to the list, in which the double gallbladder with two complete cystic ducts was removed surgically.

This anomaly is best explained by the formation of two embryonic buds from the common bile duct, instead of the usual one. These buds are originally solid cords of epithelial cells which later acquire a lumen.

REPORT OF A CASE

History.—A white woman, aged, 42, married, Jewish, had an irrelevant family history. A sister had died of tuberculosis in 1918. The patient was married at the age of 19 years; the husband and 4 children were living and well. A child had died of Pott's disease at the age of 3 years, probably contracted from the aunt. An abortion had occurred after the third pregnancy. The chief complaint was epigastric distress, jaundice and pruritus.

The patient attributed her present condition to two factors: 1. Four months before we saw her, she was dined and wined excessively on a trip to the east. No unusual digestive disturbances occurred at the time. 2. Two months before we saw her, she was in an automobile collision. There were no definite injuries, but she was badly shaken up and spent two weeks in the hospital. During this time, she had an attack of severe cramping pain in the epigastrium, with radiation around the right costal margin and through to the back. There was residual soreness. She then had repeated similar attacks, which usually followed automobile trips, but sometimes occurred at night. She belched frequently, was afraid to eat, and sometimes induced vomiting to obtain relief. Jaundice began three weeks before she consulted us, and had persisted. Intense general pruritus had annoyed her for over two weeks.

She had had an attack of similar pain followed by jaundice two and one-half years previously. Since that time she had been unable to eat veal and pickles. There had been minor attacks of indigestion but no real pain until the present illness. During her pregnancies she was troubled with heartburn but did not have serious difficulty.

The patient had had measles and pertussis in childhood. She had had tonsillitis frequently previous to tonsillectomy in 1915. Influenza, in 1918, was followed by inflammatory rheumatism. Operative repair of a rectocele was done in 1929.

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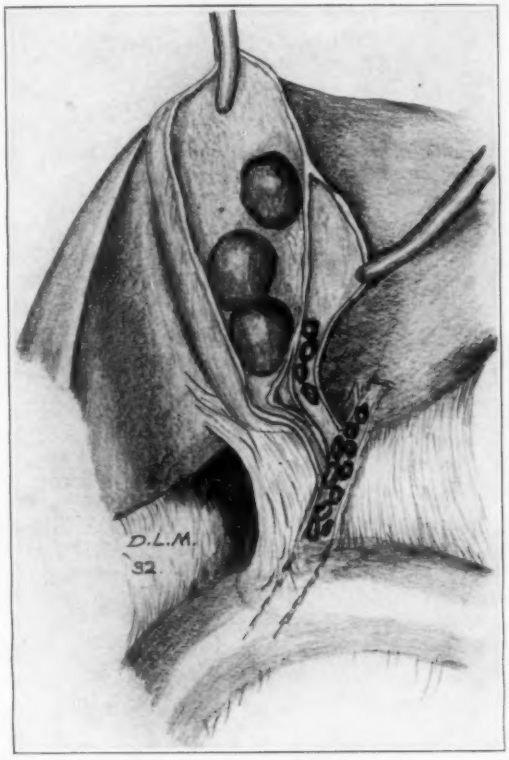
9. Nichols, B. H.: Double Gall-Bladder, Radiology **6**:255, 1926.

10. Climan, M.: Duplication of Gall-Bladder Demonstrated by Cholecystography, M. J. & Rec. **130**:73, 1929.

11. Hayes, R.: Double Gall-Bladder with Double Cystic Duct, Radiology **16**: 56, 1931.

12. Cave, P.: Two Cases of Double Gall-Bladder, Lancet **1**:751, 1931.

Physical Examination.—Examination revealed a deeply jaundiced, obese woman, whose height was 63 inches (190 cm.) and whose weight was 176 pounds (79.8 Kg.); her usual weight was 190 pounds (86.2 Kg.). The temperature was 98.2 F.; the pulse rate, 100; the respiratory rate, 20, and the blood pressure, 130 systolic and 90 diastolic. The skin and sclerae were yellow. There were many scratchmarks all over the body. Examination of the head showed no abnormality, except a moderately coated tongue. A roll of fat lay just above the clavicle. The thyroid was not palpable. The patient did not present adenopathy. The breasts were



Diagrammatic sketch showing the conditions found at operation in a case of double gallbladder of the ductular variety.

large; no lumps or tenderness was present. There were normal expansion and resonance of the chest. The heart and lungs were normal. Percussion tenderness below the right scapula was noted. There was deep tenderness in the right hypochondrium. No masses or rigidity of the abdomen was observed. The edge of the liver was not palpable. The perineum was competent. The vagina was normal and the cervix short and small. There was a normal, movable uterus. No masses were found. A large, external hemorrhoid protruded posteriorly from the anus. The extremities and the tendon reflexes were normal.

Laboratory Data.—The urine was dark because of the presence of bile. The specific gravity was 1.016. Urinalysis showed: reaction acid, albumin; sugar,

none; pus, +; granular casts, 1. Examination of the blood showed: hemoglobin, 78 per cent (13 Gm.); red cells, 4,410,000; white cells, 10,200, with polymorphonuclears, 64; lymphocytes, 29; transitionals, 4, and eosinophils, 3. The serum bilirubin was 14.7 mg. and 13.62 mg.

Duodenal drainage showed only a faint bile stain after the injection of magnesium sulphate. The coagulation time was eight minutes.

A roentgenogram, without the use of dye, did not show stones. The diagnosis was chronic cholecystitis with stone in the common duct.

Operation and Course.—During five days in the hospital, 1 drachm of calcium gluconate was given three times a day. A diet high in carbohydrate and plenty of fluids were ordered. An intravenous injection of 500 cc. of a 10 per cent solution of dextrose was given twice daily on the two days before operation.

On Aug. 13, 1932, the patient was operated on at the Tacoma General Hospital. With the patient under spinal anesthesia, a long, oblique skin incision was made from the xiphoid to the right of the umbilicus. The fascia was divided in the same line, and the right rectus muscle was retracted laterally. The peritoneum was incised, and the intestines packed away to expose the gallbladder. The gallbladder was enlarged (grade 2) and thick-walled, and contained several stones. It was seized with curved forceps, and the peritoneal bands connecting it with the duodenum were snipped with scissors. Two gauze packs were placed, and exposure of the common duct was obtained by retracting the duodenum medially. The common duct was enlarged to about 2 cm. diameter. Several stones could be palpated in it. It was seized with two Allis forceps and incised longitudinally for about 1.5 cm. Much bile escaped, which was taken care of with a suction apparatus. Ten faceted, amber-colored stones were removed with a scoop, and a probe was passed through the papilla into the duodenum. Exploration of the hepatic ducts gave negative results. A Mayo Robson no. 18 catheter was placed to reach up into the right hepatic duct, and the incision in the common duct was closed with two layers of chromic 00 sutures. The catheter was transfixed with one of these and tied into place. The gallbladder was now removed by first exposing the cystic duct, which appeared enlarged tremendously, that is, to about the size of a thumb. It was divided between clamps. The cystic artery was then located, clamped and cut. The gallbladder mass was then peeled out of its fossa. The cystic artery was ligated separately. The large cystic duct stump was closed with two layers of 00 chromic sutures. At the time it was not recognized that we were dealing with two cystic ducts, instead of one, but the structure was too wide for simple ligation. The gallbladder fossa was covered by suturing peritoneal flaps at the edges. A strip of iodoform gauze was placed next to the gallbladder fossa, and this was covered with a plain Penrose drain. A Penrose cigaret drain was placed in Morrison's pouch. The appendix was seen to be obliterated. It was not disturbed. The wound was closed in the usual manner, bringing the drains out at the lower end of the incision. The immediate postoperative condition was excellent.

The convalescence was interrupted by severe and alarming hemorrhage on the ninth day, apparently secondary to infection of the wound. There was general persistent oozing from the fatty subcutaneous layer of the wound. After transfusion, the wound was opened in its entire length to clear out pus and blood. Gauze packing soaked in an extract of blood platelets controlled the bleeding. The patient left the hospital at the end of four weeks. She has regained normal health and strength.

Pathologic Report (Dr. Martin).—The unopened gallbladder measured 8 cm. in length by 4 cm. in the greatest diameter, which was the distance from the area of the attachment to the liver to the opposite surface. It was only on section that the roughly ovoid organ was found to contain two separate cavities of unequal length and diameter, both surrounded by a common, fascial coat, and each with a duct separated from the other throughout its course in the specimen.

The larger of the two cavities measured 8 cm. in length by 7 cm. in circumference, and contained three dark, smooth, stones moderately faceted, measuring 3 cm. across. The short cystic duct leading from this cavity was slightly tortuous, and had a diameter of 2 or 3 mm. The smaller cavity measured 5 cm. in length and 4 cm. in circumference, and contained four small, dark, heavily-faceted concretions, varying in greatest diameter from 5 to 12 mm. The cystic duct from the smaller cavity was straight and large, measuring 1 cm. in diameter. The walls of both cavities were moderately thick, that of the smaller cavity being appreciably thicker than that of the larger. The gross appearance of the mucosa was that of a normal-appearing trabeculation slightly accentuated in the smaller cavity.

In addition to the gallbladder there were ten small, dark, smooth heavily-faceted stones similar in size and appearance to those seen within the smaller cavity.

Microscopic sections from the wall of the larger cavity of the gallbladder showed well developed villi covered by columnar epithelium and many glandlike projections beneath the mucosa, some extending among the muscle bundles of the muscular layer. Cellular infiltration of the mucosa was moderate and diffuse, and was most conspicuous about the deeply-penetrating glands among the muscle bundles. The muscular coat was about twice the normal thickness, and showed a diffuse, cellular infiltration among which were a few pus cells.

Sections from the wall of the smaller cavity showed a similar picture, the differences being less conspicuous mucosal villi, milder and more superficial, glandular invasion, a thicker muscular coat and a greater tendency toward perivascular, focal infiltration of lymphocytes, plasma cells and endothelial leukocytes.

It is my belief that this was a true, double gallbladder having its origin in two ductal anlagen producing ducts of greatly unequal size and two mucus-lined cavities surrounded by a common fascial coat, though each had its own muscular investment. Judging, also, by color, shape and size, it seems most probable that the stones found in the common duct were formed in the smaller cavity, from which they easily escaped through the unusually large cystic duct leading from it.

The accompanying diagrammatic sketch is self-explanatory. It shows the conditions found in this case of true double gallbladder of the ductular variety.

PYLEPHLEBITIS

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In the three and one-half year period from July 1, 1928, to Dec. 31, 1931, 4 cases of pylephlebitis were encountered at operation. Of this number, 3 were found as complications of 1,027 cases of acute appendicitis and 1 of 112 cases of acute cholecystitis. Although relatively uncommon, the dread results make this a formidable complication of acute intra-abdominal visceral disease.

Pylephlebitis may be developed in three ways: (*a*) by direct introduction of organisms into the lumens of vessels emptying into the portal vein; (*b*) by continuity from continuous organs when pyogenic organisms pass through the vessels from without, causing first phlebitis and then thrombosis; (*c*) in infections of the blood stream in which collections of micro-organisms that pass through other capillaries may settle in the liver because of the retardation of the blood stream where they multiply and then reach the portal vein and cause thrombophlebitis.

In this article we are concerned entirely with the pylephlebitis arising from the direct introduction of organisms into the lumens of vessels which empty into the portal vein, the focus of infection being either the appendix or the gallbladder. In such instances, the portal vein is found to be thrombosed and suppurative, the branches being dilated and filled either with pus or with brownish-green or greenish-black thrombus, and the periportal tissue is infiltrated with yellowish-green or slate-blue pus. The surface of the liver may show numerous small, light spots, and only after section can the relationship of these suppurative areas to the portal system be recognized. On the surface of the liver there may also be clay-colored necrotic foci and dark areas of congestion. The abscesses which develop around the portal vein spread and follow the branches of the vein, and early in the disease the hepatic lobules can still be recognized. Subsequently, however, the hepatic cells become completely destroyed, and larger coalescent abscesses occur. These lobular abscesses become confluent, and a cavity system develops, with many bulgings filled with thick pus which may have a fetid odor. The contiguous tissue frequently shows a grayish-green discoloration. The pathologic picture of the disease will be more completely described in the report of the individual cases.

From the Koster Clinic, Crown Heights Hospital.

REPORT OF CASES

CASE 1.—*History.*—C. F., a man, aged 45, was admitted to the hospital on Jan. 20, 1929, complaining of pain in the upper portion of his abdomen, fever and chills.

On January 2, the patient complained of a chilly feeling followed later by pain in the lower right side of the chest. He remained in bed for two weeks, during which time he was treated for pleurisy. His condition steadily grew worse, and he was taken to the hospital.

Examination.—This revealed a thin, undernourished, emaciated, white man, about 45 years of age, who looked acutely ill and lay in bed with his knees drawn up to his abdomen. Examination of the chest revealed a lagging on the right side, with flatness from the level of the fourth rib downward to the base posteriorly, and from the sixth rib downward anteriorly. The breath sounds were distant over this area of flatness. No râles were heard. The heart sounds were irregular in rate and rhythm; the aortic second sounds were diminished; no murmurs were heard. The apex beat was not displaced, and the heart percussed out within normal limits. There were tenderness and rigidity over the entire right side of the abdomen, but these were especially marked over the upper portion. Pressure release tenderness was referred to a point on the right side 1 inch (2.5 cm.) above and 2 inches (5 cm.) to the right of the umbilicus. No fluid was demonstrable. The spleen was palpable. The liver could not be palpated because of the rigidity, but on percussion the dulness of the liver extended downward about 4 fingerbreadths below the right costal margin. On admission, the temperature was 100 F.; the pulse rate, 124; the respiratory rate, 24, and the blood pressure, 80 systolic and 58 diastolic. Urinalysis revealed 3 plus albumin, no sugar, a few granular casts, a moderate number of pus cells and a few red cells. The hemoglobin was 70 per cent; the red blood cells numbered 3,850,000; the white blood cells, 12,100; there were 82 per cent polymorphonuclears, and 18 per cent lymphocytes.

A diagnosis of abscess of the liver was made.

Treatment and Course.—With the patient under spinal anesthesia, an aspirating needle was inserted into the subphrenic space, and pus was withdrawn. With the needle in situ, an incision was made parallel to the tenth rib on the anterior lateral surface of the lower right side of the chest. About 3 inches (7.6 cm.) of the rib was resected. The diaphragm was first sewn to the pleura in a circumferential direction around the point where the needle was inserted in order to wall off the area to be drained from the pleural cavity. The diaphragm was incised alongside the needle into the abscess cavity. The superior surface of the liver was adherent to the diaphragm all around this area. The abscessed area was about 2½ inches (6.27 cm.) in diameter and was situated on the superior surface of the liver and extended about 2 inches (5 cm.) into the substance of the liver. A gauze pack was placed all around the cavity, and a cigaret drain was inserted into the cavity after a specimen of the pus was taken for examination and culture.

The patient was given dextrose intravenously and saline by clysis. The pulse was weak and irregular. Stimulation was given, and he rallied somewhat. The next morning the patient had pulmonary edema, which progressed rapidly. The pulse became imperceptible, and the patient died soon afterward.

Postmortem Observations.—The abdomen contained a moderate amount of serohemorrhagic fluid. The appendix was bound down retrocecally and showed evidence of an acute suppurative inflammation with an abscess in the mesoappendix, all of which was covered by omentum and walled off from the general

peritoneal cavity. The gastro-intestinal tract otherwise appeared normal. There was a thrombosis of the portal vein which on being incised exuded pus. The liver presented multiple abscesses varying in size from that of a pinhead to a large abscess cavity near the superior surface about the size of a plum. The spleen was enlarged about three times its normal size and was soft and spongy. The pancreas was normal. There was no thrombosis of the splenic or the pancreatic veins. The kidneys were normal. On cross-section, the liver showed many abscesses involving the branches of the portal vein. This case was typical of a suppurative pylephlebitis following appendicitis.

A culture of the pus showed gram-negative and gram-positive diplococci.

Microscopic Appearance: The wall of the portal vein was thickened, and its regular structure was replaced by granulation tissue, only a few distorted elastic

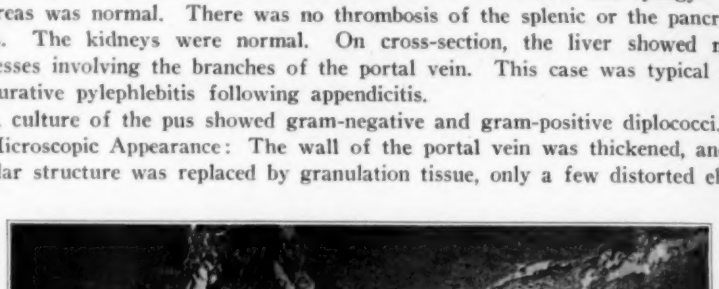

Fig. 1.—Low power magnification of a branch of the portal vein. The vein is filled with thrombus masses. The periportal connective tissue and its ramification to the adjacent hepatic tissue show cellular infiltration and edema.

fibers being left over. There was a well adherent thrombus, but parts of the thrombus were replaced by pure pus. The periportal fields throughout the liver showed considerable newly formed fibrous tissue. The walls of most of the veins were not recognizable. They were substituted by granulation tissue infiltrated with polymorphonuclears, while the lumens contained pus or necrotic masses. In the latter, huge bacterial aggregations were visualized. The hepatic tissue itself showed wide sinuses, many of which were filled with pus cells. Some foci of necrosis were seen from which the purulent infiltration seemed to radiate in all directions.

A noteworthy feature was the involvement of the lymphatics in the periportal tissue. There were numerous, considerably distended lymph vessels, some with apparently empty lumens and others filled with pus. Some of the latter showed circumscribed necrosis of their walls.

The microscopic appearance justifies the diagnosis of a subacute purulent pylephlebitis with periphlebitic lymphangitis.

CASE 2.—*History.*—A. D., a woman, aged 31, was admitted to the hospital on Aug. 28, 1931, complaining of pain in the abdomen, nausea, vomiting and general weakness. About three weeks prior to admission she had had generalized abdominal cramps accompanied by nausea and vomiting. This continued for several days, and at the end of a week the attack completely subsided, except that the patient felt weak. A few days prior to admission pains in the abdomen began again and were accompanied by nausea and chills.

The past history was irrelevant. The patient began to menstruate at 14 years of age; menstruation occurred every twenty-eight days and lasted four days. She had been married seven years. She had had no children, abortion or miscarriages. The patient had lived in Brooklyn all her life. She had no bad habits. The family history was unimportant.

Examination.—This revealed a woman, lying in bed and appearing acutely ill; the face was flushed; the lips were parched; there was sordes of the teeth. The temperature was 105.8 F.; the pulse rate, 124; the respiratory rate, 22, and the blood pressure, 124 systolic and 88 diastolic. The lungs were clear, and the heart was normal except for an increased rate. The abdomen was distended moderately; there was no rigidity or peritoneal rebound. The spleen was palpable; there was some tenderness over the edge of the liver just below the right costal margin; no fluid wave was present in the abdomen. Pelvic examination revealed a nulliparous introitus; the cervix was small, hard and closed, in the axis of the vagina; the uterus was small, firm and anteverted; the adnexa were normal. The red blood cells numbered 2,660,000; the hemoglobin was 53 per cent; the white blood cells numbered 13,400. A differential count showed: polymorphonuclears, 85 per cent; lymphocytes, 4 per cent, and monocytes, 1 per cent. The urine contained a faint trace of albumin. Chemical analysis of the blood showed 15.9 mg. of urea nitrogen, 1.3 mg. of creatinine. Van den Bergh's test gave a positive delayed reaction, both direct and indirect. The icteric index was 111; the Wassermann reaction was negative. A blood smear was negative for malarial parasites. The Widal test gave negative results.

The patient continued to have chills and a temperature ranging from 98 to 105 F., with a pulse rate varying between 104 and 140. The respiratory rate ranged between 30 and 36.

On September 1, when the temperature was 103.6 F., the pulse rate 110 and the respiratory rate 40, the patient complained of sharp pain in the lower portion of the right side of the abdomen. Examination revealed a distended abdomen with rigidity over the entire right side, but more marked in an area about 2 inches (5 cm.) to the right of the umbilicus. The condition suggested a perforation of the ileum with peritonitis.

Treatment and Course.—A laparotomy was performed under spinal anesthesia. A large amount of free fluid was found in the abdomen. The liver was studded with many abscesses containing free pus. There was a pylephlebitis. The wall of the gallbladder was thickened, edematous and contracted. The spleen was enlarged to about three times its normal size. A culture of the pus was taken. A small abscess with surrounding hepatic tissue was excised. A gauze strip was placed down to the bed of the liver, and the abdomen was hurriedly closed because of the patient's poor condition. Dextrose and insulin intravenously and saline solution by clysis were given immediately, as was also intravenous stimulation. The patient died that evening at 9:15 p. m., after becoming comatose at 6 p. m.

A culture showed gram-negative bacilli and gram-positive diplococci.

Autopsy.—When the abdomen was opened, a diffuse, fresh, fibropurulent peritionitis was found. A large collection of exudate was found between the diaphragm and the dome of the right hepatic lobe. Several loops of the small intestine were fixed in the lower part of the abdomen in the cecal region. After separating the adhesions, a small pus pocket filled with creamy greenish-yellow pus was found surrounded by dark, almost black granulations. This pus pocket was adjacent to the cecum and revealed the stump of a spontaneously amputated appendix projecting into the cavity.

Examination of the portal vein revealed occlusion by firm reddish-gray thrombus masses at the place where the mesenteric vein empties. Following the

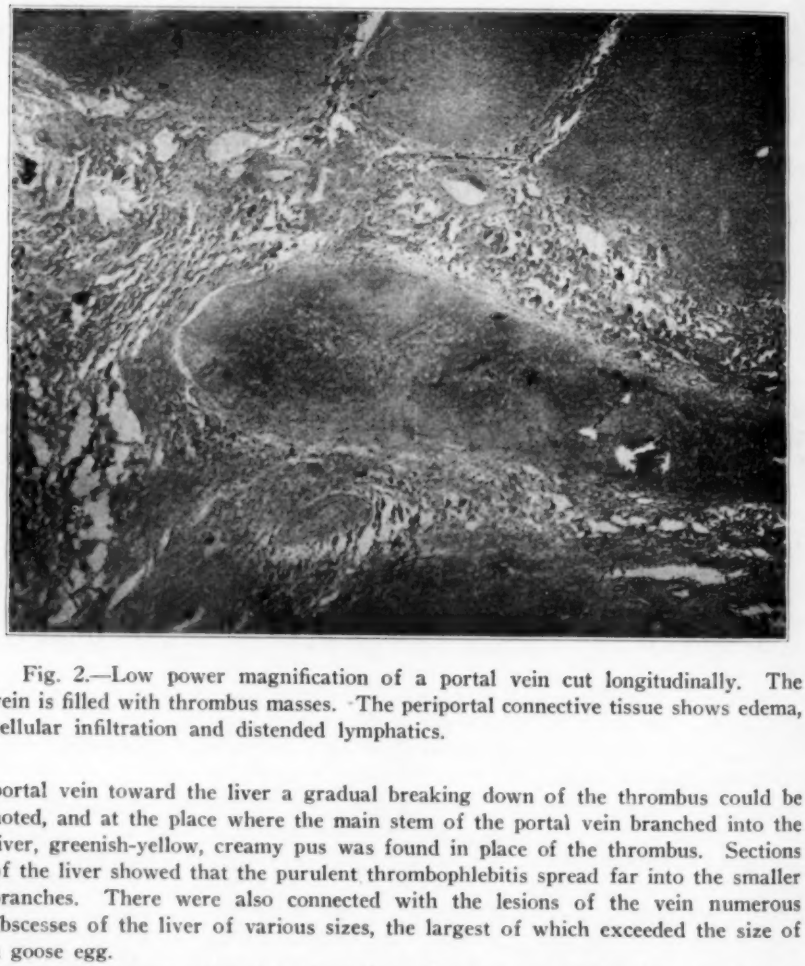

Fig. 2.—Low power magnification of a portal vein cut longitudinally. The vein is filled with thrombus masses. The periportal connective tissue shows edema, cellular infiltration and distended lymphatics.

portal vein toward the liver a gradual breaking down of the thrombus could be noted, and at the place where the main stem of the portal vein branched into the liver, greenish-yellow, creamy pus was found in place of the thrombus. Sections of the liver showed that the purulent thrombophlebitis spread far into the smaller branches. There were also connected with the lesions of the vein numerous abscesses of the liver of various sizes, the largest of which exceeded the size of a goose egg.

The spleen was enlarged to about three times its normal size. Its capsule was tense. Section showed a dark red pulp with well developed trabeculi.

Case 3.—*History.*—M. F., a woman, aged 25, married, was admitted to the hospital on Sept. 23, 1931, complaining of pain in the epigastrium and the right

lower quadrant, nausea and vomiting. Two weeks prior to admission the patient had had an attack of pain in the epigastric region accompanied by nausea and vomiting which lasted four days. Then the symptoms subsided. She had had four similar attacks within the last two years. A week prior to admission, when she began to menstruate, the attack recurred and localized itself to the right lower quadrant.

Examination.—On admission, the temperature was 105 F.; the pulse rate, 112; the respiratory rate, 28, and the blood pressure, 120 systolic and 70 diastolic. Examination revealed an adult female patient lying in bed, appearing acutely ill; the face was flushed. There was a noticeable icteric tint to the sclera. There were a few fine crepitant râles in the right side of the chest posteriorly. The heart sounds were regular but rapid. No murmurs were heard, and the heart was not enlarged. The abdomen was tender in the right upper and right lower quadrants, and was somewhat distended. There was spasticity of the entire right rectus muscle, which was more marked over McBurney's point. There was no rebound tenderness. The spleen was easily palpable. A vaginal examination revealed a multiparous introitus; the cervix was in the normal axis and closed; the uterus was in the normal position. The left adnexa were normal, but the right fornix presented a sense of resistance and tenderness. The red blood cells numbered 4,530,000; the hemoglobin was 90 per cent; the white blood cells numbered 15,200. A differential count showed: polymorphonuclears, 89 per cent; lymphocytes, 9 per cent, monocytes, 1 per cent, and eosinophils, 1 per cent. Urinalysis was entirely negative. Pancreatic ferment tests gave negative results. The blood sugar was 116 mg. and the urine negative before a sugar tolerance test, which showed: blood sugar, 160 mg. and urine negative at the end of half an hour; blood sugar, 228.6 mg. and urine 9 per cent sugar at the end of one and one-half hours; blood sugar, 173.8 mg. and urine 3 per cent sugar at the end of two hours. The icteric index was 19. The reaction to the direct van den Bergh test was positive.

A diagnosis of a suppurative pylephlebitis following an appendical inflammation and accompanied by cholecystitis was made.

Treatment and Course.—On September 24, an exploratory laparotomy was performed under spinal anesthesia. There was a slight amount of free serous fluid. The gallbladder was normal except for edema and inflammation of the ampulla and the cystic duct. No calculi were found; the edge of the liver was round. The entire pancreas was normal. The spleen was slightly enlarged and beefy. The portal vein was markedly edematous, injected and thickened. The uterus and adnexa were normal. The major portion of the appendix was hidden by a mass of adhesions formed by contiguous cęcum and omentum. The omentum was markedly injected and bled easily on separation, which brought to view an acutely inflamed appendix with small areas of necrosis on it and an abscess in the meso-appendix. No fat necrosis was present. The entire abdominal viscera were inspected. The appendix was removed by electrocautery after ligating and cutting the meso-appendix. The stump was inverted into the cecum by means of a purse-string suture. A gauze strip was inserted into the appendicular area through a stab wound to the right of the right rectus incision. The abdomen was closed in layers.

After operation, dextrose and insulin were given intravenously and saline solution by clysis. The abdominal wound drained profusely. On September 26 (the second day after operation), the patient suddenly became cyanotic and dyspneic and began to perspire profusely. The temperature dropped to 100 F., and the pulse rate to 110 a minute. The respiratory rate increased to 36 per minute.

Examination revealed a marked dilatation of the stomach, which was relieved by lavage. Examination of the chest was negative. The administration of dextrose intravenously was continued, the patient being unable to take anything by mouth because of nausea. On September 29, drainage from the abdominal wound was profuse; the temperature was 102.4 F.; the pulse rate, 130, and the respiratory rate, 32. The abdomen was distended, and the patient was nauseated. An enema produced fair results. A gastric lavage was given, with fair results. On September 30, a transfusion of 250 cc. of whole blood was given by the Koster method. A cannula was sewn into the vein in order to have a continuous intravenous infusion of 10 per cent dextrose in physiologic solution of sodium

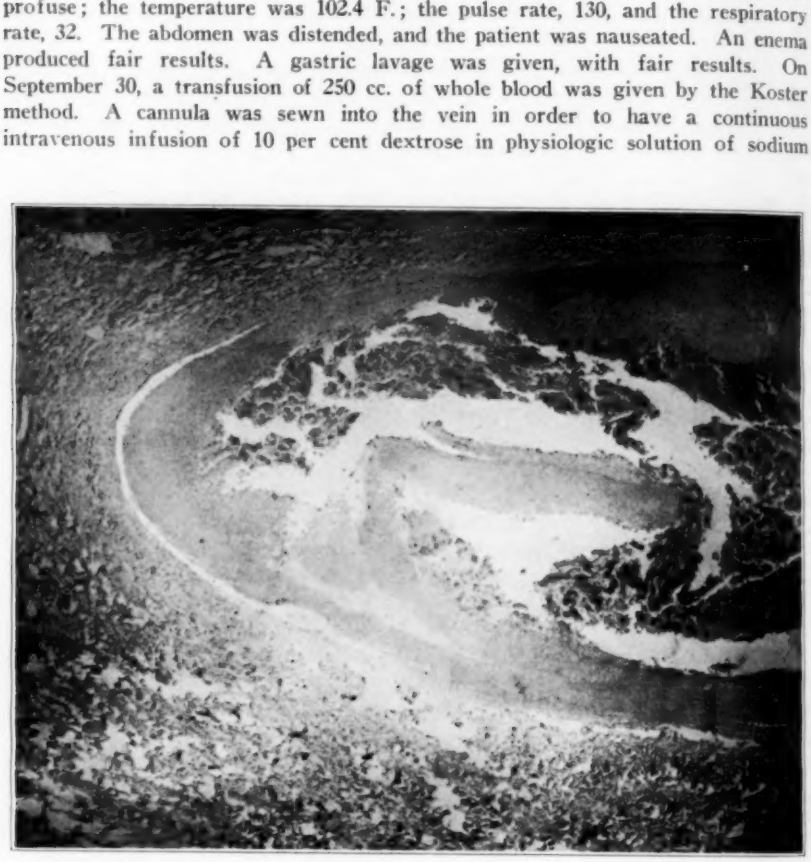

Fig. 3.—Cross-section of a large intrahepatic branch of the portal vein. The vessel wall shows cellular infiltration and is hardly distinguishable from the surrounding periportal tissue. Remnants of hyaline thrombus material form a band lining the wall of the vessel. Inside a new lumen has formed by suppuration and is partly filled with masses of leukocytes.

chloride. The temperature was 102.4 F.; the pulse rate, 160, and the respiratory rate, 44. The patient's condition was becoming more critical. The abdomen was distended again, and the drainage was marked. The pulse rate continued to rise steadily in spite of various medications, such as digifolin. The temperature rose to 105.4 F. The patient died at 5:30 p. m. the same day.

Postmortem Observations.—As the abdomen was opened there was a gush of serohemorrhagic, purulent fluid. The small intestine and the stomach were markedly distended up to about 1½ feet (45.72 cm.) from the ileocecal junction,

where the remaining part was kinked up by adhesions, causing an obstruction. A slough was present around the lateral pelvic and the abdominal wall where the appendix had been removed. The spleen was about three times the normal size. The pancreas was normal. On cross-section, both the spleen and the pancreas appeared normal. There was no thrombosis of the splenic or the pancreatic veins. The liver seemed normal in size, but on cross-section there were many areas of abscess formation around the portal triads. The portal vein was thrombotic and suppurative for about 3 inches (7.6 cm.) from its entrance into the liver. The kidneys were normal.

Microscopic Appearance: The wall of the portal vein was surrounded by ample, well vascularized and edematous granulation tissue. The wall proper was also edematous; the fibrils were in loose arrangement, and most of the elastic tissue was broken up (as shown by special stains). Closely attached to the intima was a thrombus consisting mostly of fibrin which was canalized, the lumen being filled with pus. Examination of the liver proper showed many small and large abscesses which originated from the branches of the portal vein. In most places the portal vein was completely destroyed. The abscesses were walled off only by a layer of large phagocytes and outside of this by a narrow zone of granulation tissue. The relationship of these abscesses to the portal vein was brought out by the presence of bile ducts adjacent to the abscess or by remnants of a thrombus in the periphery of the abscess cavity. In many areas free from abscess there were infiltrations of the periportal fields with round cells and leukocytes and occasionally quite massive granulation tissue. These findings indicated a subacute character of the pylephlebitic lesion.

Case 4.—*History.*—F. R., a woman, aged 47, was admitted to the hospital on June 25, 1929, complaining of chills, fever and sweats. Chills began four weeks prior to admission and were followed by high fever and sweats. This syndrome recurred daily. She had lost 20 pounds (9 Kg.) in the last three weeks.

The past history revealed that for three years the patient had had some vague abdominal distress referable to the right upper quadrant and the epigastrium. The patient had been treated for pyelitis about thirteen years previously. She was married twenty-six years prior to admission, and had four children who were living and well. Her habits were good. The family history was irrelevant.

Examination.—This revealed an undernourished adult, who appeared acutely ill. The temperature was 101.6 F.; the pulse rate, 120; the respiratory rate, 24, and the blood pressure, 120 systolic and 66 diastolic. Examination of the chest revealed equal expansion on both sides; the breath sounds were normal; there were no râles. The heart was not enlarged; the apex beat was normal but slightly rapid, and there were no murmurs. The abdomen presented tenderness in the right upper quadrant and the epigastrium. The edge of the liver was palpable about 2 fingerbreadths below the right costal margin. The kidneys were not palpable. No other points of tenderness or masses were palpable in the abdomen except for a slightly enlarged spleen.

Urinalysis showed a trace of albumin and a few white blood cells, but was negative for sugar. The hemoglobin was 62 per cent; the red blood cells numbered 3,370,000; the white blood cells, 8,000. A differential count showed: polymorphonuclears, 64 per cent; lymphocytes, 32 per cent; monocytes, 2 per cent, and eosinophils, 2 per cent. A blood culture and stool culture were taken, but both were reported negative after seventy-two hours. A blood smear taken for malarial parasites was negative. Chemical analysis of the blood showed: urea nitrogen, 14 mg.; creatinine, 1.5 mg.; uric acid, 2.72 mg.; dextrose, 117.6 mg., and carbon dioxide, 64 mg.

A diagnosis of abscess of the liver or pylephlebitis was made.

The patient was given dextrose intravenously and saline solution by clysis. Fluids were forced, and alcohol sponge baths were given for fever when necessary. On June 26, severe chills occurred which lasted thirty-five minutes, and the temperature rose to 106.2 F. after the chills. The pulse rate was 120; the respiratory rate, 24. A blood smear and blood culture were taken during the chills, both of which were reported as negative. The patient continued to have chills, fever and sweats until July 1, when a roentgenogram of the chest and the upper part of the abdomen showed a slight elevation of the right side of the diaphragm with limitation of motion and suggested abscess of the liver.

Treatment and Course.—On July 2, a laparotomy was performed under spinal anesthesia. The findings included many old adhesions of the gallbladder and omentum. The wall of the gallbladder was thickened and distended and contained a purulent fluid (empyema). There were thrombosis and varicosities of the cystic vein. On cutting into the cystic vein, free pus was demonstrated. The dome of the liver was free and not adherent to the diaphragm. Small multiple abscesses of the liver were found involving all the lobes. Free serous fluid was found in the abdominal cavity. The appendix was retrocecally bound down by one old adhesion to the cecum, but was not inflamed. The spleen was enlarged to about twice its normal size. The wall of the portal vein was edematous.

The procedure was to cauterize all visible abscesses with the electrocautery. The gallbladder was removed after the cystic duct and vessels were ligated and cut. One gauze strip was placed in the bed of the gallbladder. Two other gauze strips were used to wall off the larger abscessed cavities. A specimen of the pus from the abscess of the liver was taken for examination and culture. The abdomen was closed in layers.

As soon as the patient was put to bed, dextrose intravenously with insulin and saline solution by clysis were given. Stimulation was given because of the poor quality of the pulse. Early the next morning the patient's condition was poor. Only one heart sound was heard at the apex, and pulmonary edema was setting in. The patient died at 11:30 a. m.

A smear of pus and a culture revealed gram-positive cocci.

Postmortem Observations.—The gastro-intestinal tract was apparently normal. The spleen was twice the normal size. The kidneys and pancreas were normal. There was no thrombosis of the splenic or the mesenteric veins. Examination of the liver showed absence of the gallbladder, with the cystic vessels and the cystic duct separately ligated with catgut. There was a purulent thrombophlebitis involving the cystic veins and extending by means of the portal vein into the liver and resulting in multiple abscesses of the liver at the portal triads. Some of the abscesses of the liver had been charred by means of the electrocautery. On cross-section, the liver showed multiple abscesses varying in size from that of a pinhead to that of a large pea.

Microscopic examination was similar to that in case 3. The pathologic report on the gallbladder was acute cholecystitis.

COMMENT

Pylephlebitis and abscess of the liver cannot be considered as synonymous; the latter is almost invariably the sequel to the former, but not all abscesses of the liver result from suppurative inflammation in the portal vein. The abscess may arise through four channels; the

portal veins, the hepatic artery, the bile ducts and possibly the lymphatics. It is only when the infection travels by way of the portal veins that there can be both pylephlebitis and hepatic abscesses, and even then the two need not be associated. If the abscess is single and the result of septic extension from the appendical veins, it is almost always found in the right lobe of the liver.

Serege [1] injected Chinese ink into the splenic veins of dogs and found particles only in the left lobe of the liver. When the injection was made into the large mesenteric vein, particles were found only in the right lobe. These findings were confirmed by Glenard; [2] however, Bauer [3] and others were unable to distinguish any difference in the distribution of the ink after injection into the splenic and the mesenteric veins, and they discredited the hypothesis of Serege and Glenard that a dual current existed in the portal veins. Bartlett, Corper and Long,[4] on examining microscopic sections of lobes of dog's liver after the injection of olive oil into the tributaries of the portal veins, came to the conclusion that there was a dual current and that blood from the stomach, spleen and duodenum and the first portion of the jejunum and rectum flowed mainly to the left lobe, while blood from the lower part of the jejunum, ileum and first portion of the large intestine flowed mainly to the right lobe. Dick [5] investigated the stream-line phenomena; he identified and studied the currents of the portal vein by placing a powerful electric light behind the vessel, thus satisfactorily transilluminating the moving blood currents. He was in this way able to demonstrate sharply segregated portal currents. The position of the currents varied with the tributary into which an injection was made; for example, when an injection was made into the splenic vein, the dye-stained blood was seen to enter the left side of the portal vein in a narrow stream, preserving a narrow ribbon-like course throughout the entire length of the portal vein. When dye was injected into one of the jejunal veins, a narrow abruptly demarcated stream was observed maintaining a constant undeviated course along the right wall of the portal vein. Following the dye into the liver he found, with few exceptions, that it was carried to fairly constant and definite areas in the liver from the various tributaries of the portal vein, which he used as the injection site. When the dye was injected into the splenic and gastric veins, it was conveyed to the left half of the liver almost entirely. Dye introduced into the veins of the upper part of the duodenum, the

1. Serege, H.: J. de méd. de Bordeaux **31**:271, 1901.
2. Glenard, F.: Bull. et mém. Soc. méd. d. hôp. de Paris **3**:18 and 386, 1901.
3. Bauer, A.: J. de l'anat. et physiol. **45**:1, 1909.
4. Bartlett, F. K.; Corper, H. J., and Long, E. R.: Am. J. Physiol. **35**:36, 1914.
5. Dick, B. M.: Edinburgh M. J. **35**:533, 1928.

head of the pancreas and the jejunum was carried almost exclusively to the right lateral lobes of the liver. When injections were made into the colonic veins, the dye was distributed to all parts of the liver, but more particularly to the large lobe of the left side. In the instances in which the colonic veins did not form a common trunk, but had a separate combination in the large mesenteric veins, it was found that injection into the veins of the proximal colon produced a freer distribution of dye in the right half of the liver. These experiments are cited to offer a possible explanation for the greater frequency of right-sided solitary abscess of the liver. If the abscesses are multiple, they are distributed along the portal system, and the final picture is the resultant of forces tending toward segregation of abscesses on the one hand and toward confluence on the other.

In a review of the literature it is noticeable that there is a looseness in the use of the terms pylephlebitis and pyemic abscesses of the liver which is not only unwarranted, but also decidedly misleading. This is particularly true in quoting statistics of incidence and mortality. Thus it is possible to have pyemic abscesses resulting from invasion through the hepatic artery, in which instance no pylephlebitis is demonstrable. It can also be readily seen that there might be great differences in the results of treatment of abscesses of the liver following pylephlebitis and abscess of the liver due to dysentery.

Since Waller's [6] report of a case following appendicitis, there may have been many statistical contributions on the incidence of pylephlebitis. In 1886, Fitz [7] found the complication at autopsy in 11 of 257 cases of appendicitis. In 1897, Armstrong's [8] postmortem observations in 546 deaths for appendicitis showed pylephlebitis in more than 5 per cent. In 1903, Gerster [9] reported 1,189 operative cases of acute appendicitis in which pylephlebitis was a complication 9 times. This embraced his own experience from 1892 to 1901, at Mount Sinai Hospital, New York. In 1905, Munro [10] reported 15 undoubted cases. In the same year, Rendle Short [11] collected 1,000 cases of acute appendicitis with 4 instances of complicating pylephlebitis. In 1907, A. and E. Moschcowitz [12] reported 7 instances in 1,529 cases of appendicitis. In 1909, Giertz [13] published 4 cases occurring in 533 cases of appendicitis. In

6. Waller, quoted by Babler, E. A.: Ann. Surg. **61**:589, 1915.
7. Fitz, H. R.: Am. J. M. Sc. **92**:321, 1886.
8. Armstrong, G. E.: Brit. M. J. **2**:945, 1897.
9. Gerster, A. G.: M. Rec. **63**:1005, 1903.
10. Munro, J. C.: Ann. Surg. **42**:692, 1905.
11. Short, A. R.: Index of Prognosis, New York, William Wood & Company, 1918, p. 84.
12. Moschcowitz, A., and Moschcowitz, E.: Arch. f. klin. Chir. **3**:82, 1907.
13. Giertz, K. H.: Ueber akute eitrige Wurmfortsatzperitonitis, Munich, J. F. Bergmann, 1909.

1913, Braun [14] found it 8 times in 600 cases of appendicitis. Petrén [15] found, during 1,340 autopsies after acute appendicitis, pylephlebitis in 5 per cent.

Colp,[16] reviewing all cases of appendicitis at Mount Sinai Hospital from 1916 to 1925, found that in 2,841 cases there were 9 instances of complicating pylephlebitis. In our 1,027 cases of acute appendicitis, we found pylephlebitis in 3 instances. Other statistical studies have been omitted because careful perusal and analysis reveal the indiscriminate grouping of the pylephlebitis with abscess of the liver of origin other than in infection of the portal vein.

Many cases of solitary abscess of the liver following appendicular inflammation have been reported. These are related to pylephlebitis only as the pathogenesis depends on a thrombophlebitis of the appendicular vein with the detachment of a septic embolus from a thrombus, its migration via the superior mesenteric and portal vein and lodgment in the liver with a resultant abscess. Or, as Quenu and Mathieu [17] insist, any aseptic embolus may travel the same route, reach the liver and produce a focus of necrosis, which is then capable of becoming infected by blood-borne organisms, but fundamentally the pathologic processes are different.

Thus, the important single cause of the condition is suppurative appendicitis. Langdon Brown [18] found acute appendicitis responsible for 42 per cent of 46 collected cases of pylephlebitis. However, infection in any organ the venous return from which empties into the portal vein may produce the complication.

One of the most characteristic signs of the disease is the change in temperature. Chills accompanied by a rapid rise in temperature, occurring in the progress of an acute inflammation of any organ in the abdominal cavity the veins from which drain into the portal system, must always be considered significant of entrance of septic material into the general circulation, after it passes through the liver. If it occurs before operation, it helps in arriving at a complete diagnosis, and if it occurs after operation it points toward a serious complication. The variations in temperature may occupy a wide range, from slight fluctuations between 101 or 102 F. to marked variations from 98 to 99 F. to 104, 105 or 106 F., as can be seen by comparing the fluctuations in case 2 with the changes in temperature in the other cases. The degree of variations or the degree of change in temperature daily is no indi-

14. Braun, H.: Beitr. z. klin. Chir. **86**:314, 1913.
15. Petrén, G.: Beitr. z. klin. Chir. **94**:225, 1914.
16. Colp, R.: Surg., Gynec. & Obst. **43**:627, 1926.
17. Quenu, E., and Mathieu, P.: Rev. de chir. **44**:519, 1911.
18. Brown, L.: St. Barth. Hosp. Rep. **37**:95, 1901.

cation of the number of abscesses or the diffuseness of the lesion in the liver. Profuse perspiration is a frequent accompaniment of the daily chill. The chill need not necessarily be daily. Remission in both the chill and the rise in temperature may occur even though the disease has progressed to quite a considerable degree.

Pain over the liver is not an invariable accompaniment of the lesion, and its inconstancy helps make the diagnosis more difficult. When present, it is usually dull, is located in the right upper quadrant and has extension to the shoulder blades. A review of the case reports presented in the literature shows that but little emphasis is laid on pain as a symptom of pylephlebitis.

Whereas a high leukocytosis would be expected with an infection of this kind, it does not always occur. In the 4 cases herein reported the white cell count ranged from 8,000 to 15,000. It is particularly significant that the case in which the blood count was 8,000 was one in which the pylephlebitis complicated an empyema of the gallbladder. In the differential count, the polymorphonuclears ranged from 60 to 89 per cent. Thus, the leukocyte count also cannot be considered of great diagnostic significance.

Tenderness is almost invariably present, although it may be so slight as to be considered negligible. It may frequently be found directly over the portal vein.

Jaundice is almost always present and is one of the early signs of involvement of the liver. When it appears early in the disease (before the diagnosis of appendicitis, cholecystitis, etc., is made), it may be misleading, as it may draw attention away from a causative appendicitis and direct it to the biliary passages in a case in which involvement of the latter is only the terminal complication. This occurred in case 3. In many of the reported instances the bilirubin content of the blood serum showed a progressive increase long before the jaundice could be noted clinically. The appearance of jaundice as a postoperative complication of appendicitis should arouse suspicion of the development of a complicating pylephlebitis. A slight icteric tint to the sclerae may appear even before the chill and fever.

In the description of the pathologic processes found at autopsy in cases of pylephlebitis, enlargement of the spleen is mentioned in a great many of the cases reported. In this connection it seems strange that little mention is made of splenic enlargement as a clinical sign of value in pointing toward the diagnosis. In all of our cases enlargement was noticed clinically, and it seems to us that it may be utilized as a valuable diagnostic sign. The presence of serous fluid in the peritoneal cavity in small amounts is expected because of the general peritoneal reaction to the inflammation even of an organ in direct continuity to it. With

complete obstruction of the portal vein, however, the development of ascites is far more likely to occur. This, however, is not seen except terminally. Of the other symptoms of anorexia, nausea, vomiting, lassitude and emaciation, little need be said, because they are not characteristic of this condition any more than they are of infectious conditions any place in the body. Examination of the blood for organisms is of little value in these cases, because in most instances the invading organism is Bacillus coli, which rarely breaks into the blood and if it does is quickly destroyed there.

In the typical case the diagnosis can be made by the shift of symptoms from the appendicular region to the hepatic region following repeated chills and fever, in addition to the other symptoms and signs enumerated, all occurring in the absence of manifestations of extensive peritonitis. Munro's [10] dictum that the "most important clue in making a diagnosis is the recognition of the causative appendicitis," while true in most instances, is certainly not universally applicable, as case 4 shows. It must therefore be remembered that the original focus may be in any organ in the abdomen, venous drainage from which empties into the portal system.

The treatment of this condition is primarily prophylactic, and such prophylaxis may be accomplished in two ways. There can be no doubt that if all cases of acute appendicitis were to come to operation at an earlier date through earlier recognition of the condition, there would be fewer cases of pylephlebitis. When, however, the surgeon encounters a case of appendicitis and notes in cutting the meso-appendix that there is no bleeding because of thrombosis of the appendical vein, he should consider that case as one in which pylephlebitis may develop. This is true whether there is a history of chills or not. Remembering, however, the low incidence of occurrence of this grave complication and also that thrombosis of the appendical veins is encountered quite frequently in gangrenous appendicitis and perhaps less frequently in the acute suppurative type of inflammation, the question of whether it is possible to determine in which of these cases pylephlebitis will develop presents itself. Since many acutely inflamed appendixes contain definitely thrombosed veins, and since the patients in whom they are found make uneventful recoveries after simple appendectomy, apparently because the early appendectomy removed the primary bacterial focus and also because even if bacteria are sent to the liver from the thrombosed veins they are apparently destroyed by that organ the bactericidal properties of which are potent, it becomes apparent that in the absence of more data it is impossible to predetermine in which cases complications will develop.

If at operation a frank suppurative phlebitis of the mesentery is evident, before resorting to and before performing the appendectomy a ligation or excision of the ileocolic vein is indicated. The method of procedure that may be adopted is any one of the following: Gerster [9] advocated incision and evacuation of the infected thrombosed vein. This is best done by opening the veins in the stump of the appendix and following them up on the cecum and into the ileocecal angle as far as there is suppuration, evacuating the thrombosed vessels and instituting drainage by means of gauze. Wilms [19] advocated ligation of the veins in the ileocolic angle for reasons similar to those advocated by Trendelenburg in ligating the ovarian veins in cases of puerperal pyemia. This is done by immobilizing the cecum and ascending colon from the posterior abdominal wall, exposing the veins, which are then tied without occluding the artery. Drainage is placed to the cecum. The variation in the venous return from the ileocecal angle makes it difficult to ligate all the venous channels successfully, and it must be remembered that unless all the veins at the angle are ligated the purpose of the operation may easily be defeated. Another objection to the procedure is the danger of destroying the circulation with resultant gangrene of the bowel. Braun [14] described ligation of the ileocolic vein, since this vein is the only channel through which the thrombi in the appendical vein can travel to reach the portal vein. Its ligation is a rational procedure in the attempt to arrest the bed of infection. When the infection has already spread beyond the confines of the ileocolic vein and a pylephlebitis has already developed, operative intervention is of no value unless a well defined abscess has developed, in which case the indication, of course, is for incision and drainage. The literature on the value of ligation of the portal vein in cases of pylephlebitis is clearly reviewed by Colp,[16] and the conclusion is that such an operation is probably never indicated, even though the experimental studies of Neuhof [20] and operative attempts on human beings (Beer [21]) have demonstrated that the "hepatopetal" veins in the gastrohepatic omentum in certain persons can efficiently carry on the portal circulation after complete occlusion of the portal vein by ligation. For if the vein is not yet the seat of thrombosis and suppuration, because of the small percentage of incidence of pylephlebitis, there is great likelihood of its remaining free. If it is believed that it may become infected by extension, then ligation or, preferably, resection of the ileocolic vein, a relatively simple procedure, is likely to prevent the extension. When infection of the portal vein and the intrahepatic portion of the portal

19. Wilms, M.: Zentralbl. f. Chir. **36**:1041, 1909.
20. Neuhof, H.: Surg., Gynec. & Obst. **16**:481, 1913.
21. Beer, E.: Am. J. M. Sc. **150**:548, 1915.

system has already occurred, all that can be expected of ligation of the portal vein is the demonstration of the ability of the anastomotic veins of the gastrohepatic omentum to relieve engorgement of the abdominal viscera drained by the portal system on the one hand and an interruption of the bacterial feeding process from the original focus of infection on the other. Neither of these, however, influences the outcome, because there are already enough infection and damage of the liver, and also a remaining focus for more dissemination in that portion of the thrombosed and infected vein central to the ligature. Indeed, many cases have been reported in which complete thrombosis of the portal vein existed at the time of operation, and in such instances, even on theoretical grounds, ligation can scarcely be of value.

It must also be remembered that when the process has extended because ligation or resection of the ileocolic vein has not been successful in preventing the spread from the primary thrombus, the patient may occasionally recover.

ELECTROBASOGRAPHIC METHOD OF RECORDING GAIT

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Conditions due to neuromuscular and vascular pathologic processes together with deformities of the lower extremities are generally recognized causes of disturbances in locomotion. The treatment of these disorders embraces the fields of medicine, general surgery and orthopedics. Whenever treatment of such conditions is seriously undertaken, the difficulties accompanying the visual analysis of gait are recognized.

Efforts directed toward the development of methods for recording gait have been less numerous than the apparent need for such clinical advantage would seem to justify. Most of the studies in this country and abroad have been made with a consideration for the physiology of locomotion without reference to the possibility of such studies being of practical value to the various branches of clinical medicine.

We accept the premise that no two people walk alike. We doubt the possibility of determining a normal gait. Our position is comparable to that of Wunderlich's and others in establishing clinical thermometry. All of us accept the fact that of a hundred people who show no evidence of disease, records of their respective temperatures would not be identical. Nevertheless, 98.6 F. has become the point from which elevated and subnormal temperatures are measured. Moreover, the temperature chart is used as an index of the progress of treatment. Our efforts have likewise been directed toward the development of a method of recording gait for the purpose of providing a series of clinical records by which the progress of treatment may be expressed through the precision of physical laws. The diagnosis in terms of localization and etiology of abnormal gait must continue to depend on the facts revealed by the individual history, physical examination and laboratory data.

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All methods heretofore developed for recording gait, including those that have been published by others and ourselves, have failed to be of practical clinical value. We consider the available evidence for this statement as foreign to the subject of this paper. One of us (R. P. S.) has stated the limitations of the basograph [1] as an instrument for recording gait, and the pneumographic method [2] must now be regarded in the light of prevailing fundamental difficulties which cannot be removed to provide the simplicity required for clinical application.

In addition to providing records with ease, rapidity and economy, any method of recording gait must fulfil the following requirements: 1. The mechanism must be free from the possibility of errors which would cause changes in the character of the record produced. 2. The character of the gait record of the same patient must be constant unless the gait is altered. 3. Changes in the gait record must be indicative of changes in the patient's gait.

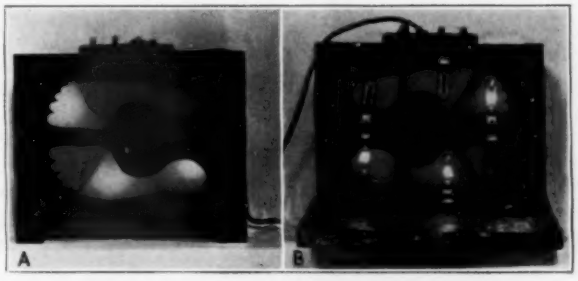

Fig. 1.—*A*, the transillumination box, front view, showing outline of feet on ground glass. *B*, the transillumination box, back view, showing lights in the three respective compartments for each foot.

A transillumination box (fig. 1) was developed in connection with the pneumographic method of recording gait. Through its use it was possible to determine the length of time that weight was borne on the heel, the head of the fifth metatarsal and the great toe of the respective feet. During locomotion weight is successively transmitted from the heel to the head of the fifth metatarsal, then to the great toe, which is last to leave the ground. When the patient walks without a limp, an equal amount of time will be spent on these respective points of the two feet. Abnormalities in gait are, therefore, the result of inequalities in time spent on the respective points of each foot, including the omission of weight-bearing on one or two of the three points on one or both feet.

1. Schwartz, R. P., and Vaeth, W. E.: A Method for Making Graphic Records of Normal and Pathologic Gaits, J. A. M. A. **90**:86 (Jan. 14) 1928.

2. Schwartz, R. P., and Heath, A. L.: The Pneumographic Method of Recording Gait, J. Bone & Joint Surg. **14**:783 (Oct.) 1932.

It is true that inequalities in pressure might be given consideration. At the present time we are in possession of evidence to support the belief that differences in the duration of time on these respective points can prevail with little or no inequality in pressure. We are of the opinion that whenever respective pressures are unequal there is an accompanying difference in the duration of weight-bearing time at that point on the respective feet. Evidence supports the belief that differences in the duration of time spent on these respective points can prevail with little or no inequality in pressure. Because of this it seems logical that a method of recording gait would be most reliable when it revealed differences in the duration of time that weight is borne on the heel, the fifth metatarsal and the great toe of the right and the left foot, respectively. The electrobasagraphic method of recording gait fulfils this requirement; it does not record pressure.

TECHNIC

The mechanism employed is as follows: Boards 42 feet (2,480.16 cm.) long and 17 inches (42.55 cm.) wide are covered with an aluminum plate. This forms the negative side of an electric circuit. On the soles of the patients' shoes (fig. 2) three-fourths inch (1.87 cm.) circular brass contacts are fastened by means of heated flake shellac. These contacts are placed under the heel, the fifth metatarsal and the great toe. The position has been determined on a mathematical basis, taking into consideration the total area of the particular shoes. For women's and children's shoes one-half inch (1.27 cm.) circular contacts are used. From these three contacts on the shoes three wires lead to a recording apparatus and the transillumination box (fig. 3).

The recording mechanism (fig. 3) consists of a one-tenth horse power direct current motor, a 48 to 1 gear reducer and a camera box in which a 100 foot roll of 70 mm. emulsion paper is driven past a horizontal slit. On the outside of the box, arranged opposite the slit, are seven 6 watt bulbs encircled by brass tubing 1 inch (2.5 cm.) in diameter. From each of these tubes another tube one-eighth inch (0.3 cm. in diameter, centered opposite the lamp filament, leads to the slit. The center light is connected to a metronome and is made to go on and off every second. This gives the timing line in terms of seconds in the middle of the paper, thereby dividing it into right and left halves. The light on the right end is connected to the contact on the right heel, the next one to the fifth metatarsal, and the third light on the right side is connected with the contact under the right great toe. The connections of the respective lights on the left side of the midline from without inward are the same: heel, fifth metatarsal and great toe. When the patient stands on the aluminum plate the lights will go on and off as these respective points of the shoes come in contact with the aluminum plate so as to close the circuit. The motor drives the paper through the camera box at a constant speed of 10½ feet (320 cm.) per minute. We are, therefore, able to record on the photographic paper, lines which represent the duration of time spent by the patient on the heel, fifth metatarsal and great toe of each foot. Moreover, we are able to determine the number of steps taken per minute.

The photographic record is developed in a dark room, as is the record produced by the electrocardiograph.

RESULTS

A typical record of the gait of an apparently normal man, aged 30, is illustrated in figure 4 *A*. The wide, broken line in the middle represents intervals of time in seconds. The lines representing the duration

Fig. 2.—Circular contacts applied to the right shoe for man, woman and child. These contacts form the positive side of an electric circuit.

Fig. 3.—Subject standing on aluminum-covered board; the wires are attached to contacts on the shoes; illumination of the right and left foot, respectively.

of weight-bearing on the heel, the fifth metatarsal and the first toe of the right and the left foot are shown on the respective sides from the edge toward the timing line.

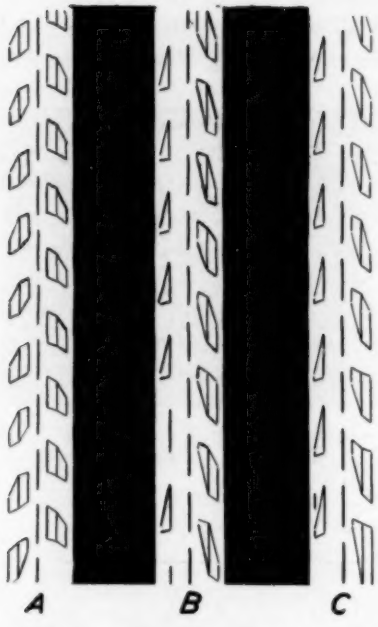

Fig. 4.—*A*, typical record of a "normal" man, H. T., aged 30, weighing 98 Kg. and 182 cm. in height. The lines representing the duration of weight-bearing on the heel, fifth metatarsal and first toe of the right and left foot, respectively, are indicated. The broad interrupted middle line indicates the time in seconds. *B*, gait record of J. S., aged 64, weighing 69.2 Kg. and 181.6 cm. in height. Lateral displacement of left scaphoid and cuboid. Note: (1) short duration of weight-bearing on left heel; (2) long duration of weight-bearing on left fifth metatarsal; (3) absence of weight-bearing on left first toe. *C*, record made on Sept. 29, 1932. Note duplication of the same patient's record made on Sept. 28, 1932 (*B*).

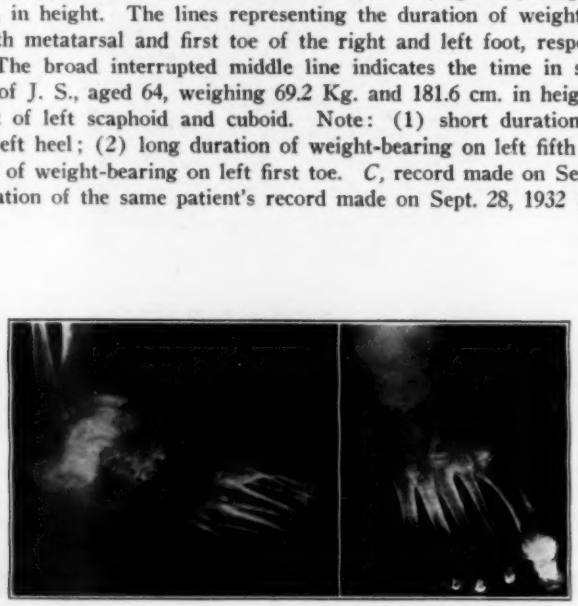

Fig. 5.—Lateral and postero-anterior roentgenograms, showing medial displacement of forefoot at the level of the scaphoid and cuboid bones.

Having thus produced a "normal" record of the foregoing character, figure 4 *B* illustrates the abnormal gait of a man who had, on May 26, 1932, suffered a medial dislocation of the scaphoid and cuboid bones of the left foot. The roentgenograms of the affected foot are illustrated in figure 5. The prevailing deformity forced the left foot in a varus position accompanied by slight equinus. Again referring to figure 4 *B*, the timing line is the same length as in figure 4 *A;* the records are otherwise very different. The unaffected right foot bore weight for a period of 1.05 seconds, and the weight was thrust in normal sequence from the heel, which bore weight for 0.783 second, to the fifth metatarsal, which bore weight for 0.729 second; and the first toe, which is normally last to leave the ground, bore weight for 0.297 second. The left foot received weight for a period of 0.786 second, 0.088 second of which was spent on the heel, while the fifth metatarsal received weight for 0.735 second with no weight being transmitted to the left great toe.

Weight-Bearing Time in Seconds

	Total	Heel	Fifth Metatarsal	First Toe
Right foot.....	1.050	0.783	0.729	0.297
Left foot.....	0.786	0.088	0.735	0.000

This record was made on Sept. 28, 1932; the contacts were removed from the shoes, and the patient was returned to the division. On the next day, the patient was sent to the gait clinic; the contacts were again fastened to the shoes according to the mathematical determinations previously made, and a second record was made (fig. 4 *C*). This is presented as an example of the evidence which, at present, indicates that we may accept the following premise: So long as the patient's condition remains unchanged, successive records of gait will reveal the same characteristics. Any method for recording gait, to provide an index of the progress of treatment, as applied by the various branches of clinical medicine and surgery, must be free from differences in records due to causes other than changes in the patient's gait.

Further evidence that the electrobasographic method of recording gait has met this fundamental requirement is presented in figure 6. On Oct. 18, 1932, a small oxygen tank fell on the patient's right second toe, producing uncomplicated trauma of the whole toe. The first record of the patient's gait was made on the day injury was incurred. Successive records were made on October 20, 24, 27 and November 3. The last record reveals complete functional recovery at a time when the subjective symptoms and physical signs of trauma had disappeared.

This series of records further indicates that the electrobasographic method does record evidence of improvement in gait when treatment favors the passing of subjective symptoms and the disappearance of physical signs of the cause of dysfunction.

Between Sept. 9, 1932, and Nov. 28, 1932, one hundred and three records were made. Seventy records were of subjects showing no visual evidence of abnormal gait, while the remaining thirty-three were of those who revealed definite departure from "normal" locomotion. A series of twelve records of gait of "normal" persons is illustrated in figure 7.

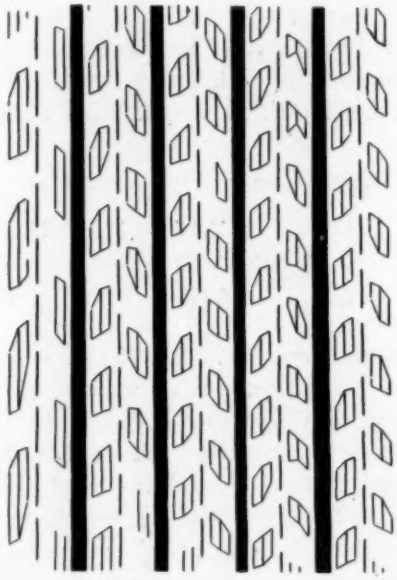

Fig. 6.—Gait records of Miss M. D., aged 33, weighing 48.5 Kg. and 161 cm. in height. Injury of right second toe with complete functional recovery as revealed by the gait records made between Oct. 18 and Nov. 3, 1932.

The detailed discussion of the interpretation of these records must receive consideration in the next paper on this subject. These twelve unselected records reveal a definite tendency toward uniformity in contour. This is particularly emphasized when comparison is made with the twelve records of gait of persons who had definite limitation in locomotion, as illustrated in figure 8. It is also evident that comparison of these records of abnormal gait, unselected for this purpose, reveals no tendency toward duplication, nor do they closely resemble the records in figure 7.

These two groups of records reveal the fact that "normal" gait may be recognized from the relationship of the lines representing the

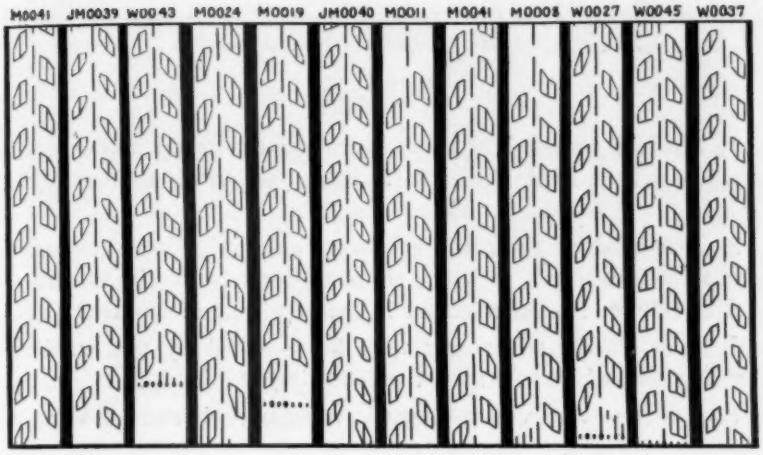

Fig. 7.—A series of twelve gait records of "normal" persons.

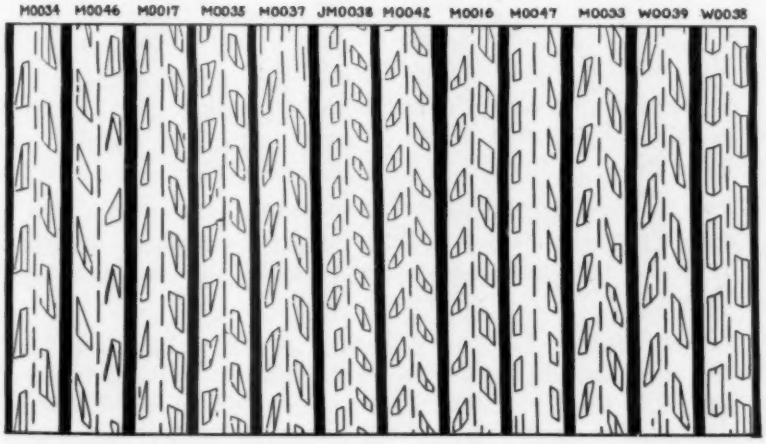

Fig. 8.—A series of twelve gait records of subjects who had definite limitations in locomotion: *M0034,* proliferative arthritis, both knees; *M0046,* degenerative arthritis; toe drop; *M0017,* medial displacement of forefoot at level of scaphoid and cuboid bones; *M0035,* right gluteus medius; *M0037,* epilepsy; *JM0038,* left equinus; *M0042,* athetosis; *M0016,* ankylosis, right hip in flexion; *M0047,* equinovarus with cavus; *M0033,* postoperative subastragalar arthrodesis for equinovarus following poliomyelitis; *W0039,* muscular dystrophy; *W0038,* muscular dystrophy.

duration of time that weight is borne on the heel, the fifth metatarsal and the great toe. Moreover, it has been demonstrated that the absence of equality in length or the omission of one or more of these lines may be accepted as evidence of "abnormal" locomotion.

SUMMARY

1. Medicine, general surgery, neurosurgery, industrial surgery and orthopedics have to do with the treatment of patients suffering from lesions which produce disturbances in gait.

2. A method of recording gait is as essential to the proper treatment of abnormal gait as is the electrocardiograph in the treatment of heart disease, or the thermometer in the recording of temperature.

3. Such clinical advantages may be provided by that method of recording gait which will meet the following conditions:

(*A*) It must assure simplicity of procedure, minimum time requirement and economy.

(*B*) The mechanism must be free from alterations in the record due to technical errors.

(*C*) Evidence must prove that changes in successive records made of the same patient's gait are due only to the way the patient walked on successive dates. Such records would then give evidence of the presence or absence of improvement under recorded conditions.

4. We have offered the premise that abnormalities in gait may be defined in terms of the duration of weight-bearing time on the heel, the fifth metatarsal and the great toe of the respective feet.

5. The electrobasographic method of recording gait photographs the duration of time that weight is borne on the heel, the fifth metatarsal and the great toe of each foot, using the patient's own shoes.

6. Data in the form of patient's records of gait have been presented to support the belief that the electrobasographic method of recording gait fulfils the provisions essential for providing the clinical advantages as outlined in 3 of the summary.

Dr. Milton Chapman, Dr. Edwin Fritz, Mr. Carson Meyer and Mr. Howard R. Patterson of the Eastman Kodak Company and Mr. F. W. James of the Bausch and Lomb Optical Company cooperated with us and assisted in this work. The continued interest of Dr. John J. Morton and Dr. Wallace Fenn has made possible the presentation of this work.

HEALING OF FRACTURES AND BONE DEFECTS AFTER VENOUS STASIS

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In this paper we shall report a series of observations on the healing of fractures or of defects in both ulnae of the dog when the veins draining one foreleg are ligated, thus creating marked venous stasis in that leg.

REVIEW OF THE LITERATURE

The literature on the effect of venous stasis on osteogenesis has been reviewed by Pearse and Morton,[1] who trace the use of hyperemia from the time of Ambroise Paré. The clinical observations in the literature stimulated these authors to study the subject experimentally. They created a small defect in the fibula of each hind leg and ligated the right popliteal vein in twelve dogs. The ligation of this vein caused venous stasis in the region of the experimental bone defect in that fibula. In eleven of the twelve dogs, union was accelerated on the side of the ligation; this was manifested by the earlier formation of callus, the earlier calcification of callus and the earlier union. In addition to the experimental work, Pearse and Morton reported two clinical cases of delayed union in which venous stasis (hyperemia) instituted by elastic compression resulted in union.

In a previous paper, Morton and Stabbins[2] reported observations made on twelve rats and three dogs in which 0.5 cm. of each fibula was removed and the left saphenous vein was ligated. In these animals nonunion occurred on the ligated side. The authors concluded that a partial block of the veins of a limb in which there is a fracture or a resection causes delay in the union. After this paper was published, one of us (Dr. Key) repeated the experiments of Morton and Stabbins on the fibulae of dogs, but the results were inconclusive and were not published. After the publication of the second paper, in which an opposite result was reported following a similar operative procedure, we felt that the observations should be confirmed. If it is true that venous

From the Department of Surgery, Washington University School of Medicine.

1. Pearse, H. E., and Morton, J. J.: The Stimulation of Bone Growth by Venous Stasis, J. Bone & Joint Surg. **12**:97, 1930.

2. Morton, J. J., and Stabbins, S. J.: Circulatory Factors Influencing Normal Osteogenesis, Ann. Surg. **86**:430, 1927.

stasis stimulates osteogenesis and accelerates the union of bone, venous stasis constitutes, so far as we know, the only method yet discovered by which this can be accomplished.

Consequently, we resected 0.4 cm. of each fibula and ligated the popliteal vein in six dogs. Ten weeks later the animals were killed, and the fibulae were removed and studied. In one animal there was slightly firmer union on the right or ligated side, and in five animals the amount of callus and degree of union were practically the same on the control and on the ligated side.

From the aforementioned observations it is evident that we were not able to confirm the results either of Morton and Stabbins [2] or of Pearse and Morton.[1] We thought that the conflicting results might be explained by an unhappy choice of site for the bone defect. Since the fibula is a small bone and is largely covered by muscle attachments, it is difficult to perform a series of uniform subperiosteal resections on it. Consequently, we decided to repeat the experiment on a larger bone, and we chose the ulna because other experimental work had shown us that this is a bone of considerable size, and that since it is splinted by the radius, any desired amount can be resected with relatively little disability to the animal.

MATERIAL AND METHODS

The material consisted of twenty-four adult dogs. Twelve of the dogs were subjected to bilateral subperiosteal osteotomy of the ulna, the bone being exposed under aseptic conditions and cut with an osteotome and the wound sutured. In six of these dogs the ligation of the veins draining the right foreleg was carried out at the time of the operation on the ulna, and in the other six the unilateral venous ligation was carried out one week later.

The other twelve dogs were subjected to operations in which a piece of bone approximately 0.5 cm. in length was resected from the shaft of each ulna. The operation was the same as the preceding one, except that instead of the osteotome the sharp bone-cutting forceps were used. After the shaft of the ulna had been exposed subperiosteally approximately 0.5 cm. of the shaft was removed with the bone-cutting forceps, care being taken to remove the entire thickness of the shaft and to make the defects in the ulnae of a given animal as nearly the same size as possible. In six of these animals the unilateral venous ligation was carried out simultaneously with the resection of the ulna, and in the remaining six the ligation of the veins was performed one week after the osteotomy.

In ligating the veins an incision was made first over the lower third of the brachial artery, and the deep veins accompanying this artery were tied and cut. Likewise, any other veins which could be found in this vicinity were tied and cut. This ligation caused congestion in the foreleg. Then a rather long incision was made in the bend of the elbow, and the large superficial veins which cross the elbow joint were ligated. Finally, a small incision was made in the vicinity of the olecranon, and a superficial vein which was usually present in this region was ligated. We thus performed ligation of all of the large veins which could be identified in the region of the elbow. In each instance the vein was doubly ligated and cut between the ligatures.

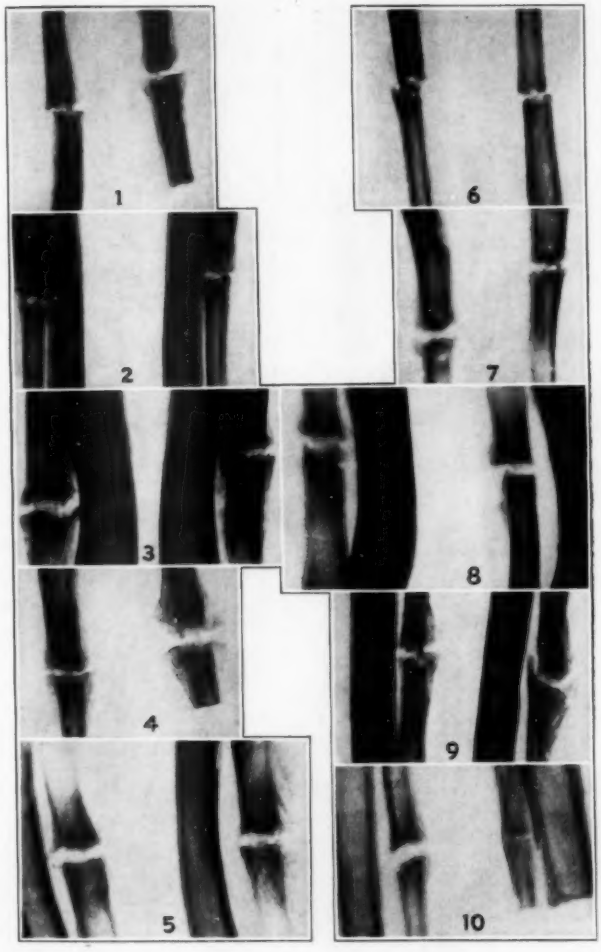

Fig. 1 (parts *1* to *10*, inclusive.—Roentgenograms of the ulnae of dogs after osteotomy. In each instance the veins draining the right foreleg were ligated at the elbow. In parts *1* to *5*, inclusive, the ligations were performed at the time of the osteotomy, and in parts *6* to *10*, inclusive, the ligations were performed seven days after the osteotomy. In each instance the x-ray photograph of the ulna of the ligated limb is on the left and the control is on the right. Pictures *1* and *6* show the condition after four weeks; *2* and *7*, after five weeks; *3* and *8* after six weeks; *4* and *9* after eight weeks; *5* and *10* after ten weeks.

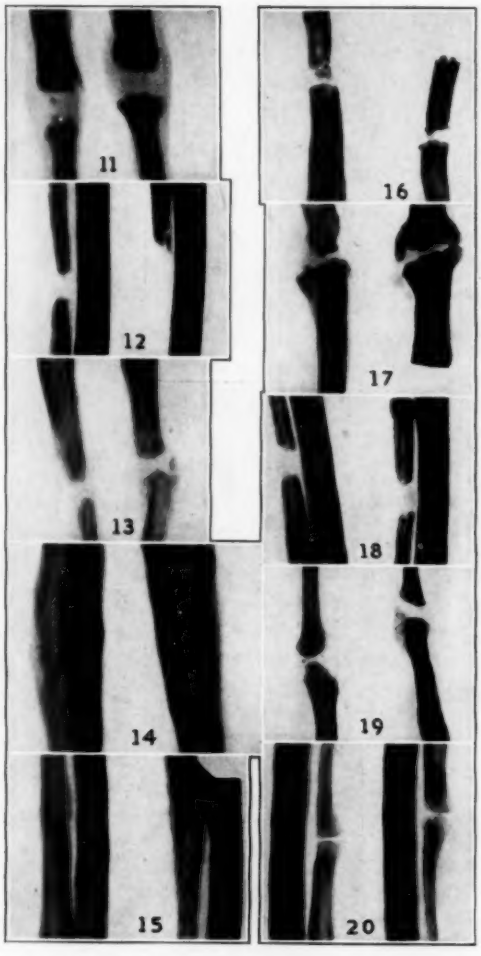

Fig. 2 (parts *11* to *20*, inclusive).—Roentgenograms of operative defects in the ulnae of dogs in which the veins draining the right foreleg had been ligated at the level of the elbow. In parts *11* to *15*, inclusive, the ligations were performed at the time the defects were made, while in parts *16* to *20*, inclusive, the ligations were performed one week later. In each instance the ulna of the ligated leg is on the left and the control is on the right. Roentgenograms *11* and *16* were taken after four weeks; *12* and *17*, after five weeks; *13* and *18*, after seven weeks; *14* and *19*, after nine weeks; *15* and *19*, after twelve weeks.

The operation on the veins always resulted in marked venous congestion of the foreleg and foot. The swelling persisted over a period of about two weeks and then decreased gradually as the circulation became adjusted. Our object in delaying the ligation of the veins until one week after the osteotomy in half of our animals was to rule out the possibility of accelerated union resulting from the presence of an unusually large hematoma at the site of the fracture or bone defect.

The animals in which osteotomies had been performed were killed at intervals of from three to ten weeks, and those with bone defects, at intervals of from three to twelve weeks. The ulnae on both sides were removed and examined and fixed in a diluted solution of formaldehyde (1:10), U. S. P. Roentgenograms of the specimens were made, and the specimens were then decalcified, sectioned, stained and studied microscopically.

RESULTS

The gross specimens disclosed no demonstrable difference in degree of union between the ulna of the leg which had been subjected to ligation of the veins and the control on the other side of the animal. Nor was any difference shown in the roentgenograms (fig. 1, *1* to *10*) or on microscopic examination. In some instances union was more advanced than in other experiments of the same or of longer duration, but this was due to the fact that the dogs were young ones; in these (fig. 2, *14* and *15*) there was approximately the same degree of union on both the ligated and the control side. There was no more bone atrophy on the ligated than on the control side.

On microscopic examination engorged, apparently dilated blood vessels were seen in the region of the fracture or defect on the ligated side, but study of the sections from the fracture or defect on the control side of the same animal revealed a similar condition.

COMMENT

In a recent paper Pearse and Morton [3] reconsidered their first opinion that blocking of the venous circulation tends to cause nonunion. They pointed out that their first operations were not carefully performed, and that the ligation of the saphenous vein was probably not effective in creating venous stasis in the region of the fracture. They further noted that ligation of the small saphenous veins has no effect on the rate of union in the fibula, while resection of the femoral artery and its branches tends to cause delayed union.

It is difficult to correlate the results of Pearse and Morton [3] with those obtained in the experiments reported in this paper. There are possible sources of error in each method. In our work it is possible that, in spite of the extensive ligation and severance of all of the veins

3. Pearse, H. E., and Morton, J. J.: The Influence of Alterations in the Circulation on the Repair of Bone, J. Bone & Joint Surg. **13**:68, 1931.

sufficiently large to be identified in the region of the elbow, the venous stasis was neither of sufficient degree nor of sufficiently long duration to influence the repair of the ulnar fractures. On the other hand, there was no question but that a marked degree of venous stasis was obtained; this lasted over a period of about two weeks, and then the limb gradually returned to a condition resembling that on the control side. The possible error in the experiments of Pearse and Morton is in the fact that the fibula is a small bone the surface of which is largely covered by muscle attachments. In our experience, conclusions drawn from experimental fractures or resections on this bone have not been reliable.

In regard to the clinical cases reported in the literature and those of Pearse and Morton in which fractures (with delayed union) united after passive hyperemia was instituted, we may state that we have had similar experiences. After reading Pearse and Morton's paper we used the method clinically and obtained union. But we are not in a position to state that the union was due to the hyperemia.

CONCLUSIONS

Ligation of the veins around the elbow caused venous congestion in the foreleg and foot of the dog, but had no effect on the healing of a fracture or of a defect in the ulna of this leg.

RIBS OVERLYING EMPYEMA CAVITIES

PATHOLOGIC STUDY

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OMAHA

Transformation of the external form of ribs overlying empyema cavities occurs so constantly that the nature of the transformation and its pathogenesis seemed worthy of investigation. An extensive search of the literature failed to provide information dealing with the subject, with the exception of descriptions of the gross deformities of ribs (Hedblom [1]).

These ribs, as every surgeon engaged in thoracic surgery has frequently observed, have lost their normal contour and have become relatively round, triangular or rectangular and somewhat larger in cross-sectional dimensions. As illustrated in figure 2 *A*, these changes have resulted from the deposition of subperiosteal new bone on the inferior borders and internal surfaces of the ribs. The greatest deposits are laid down along the angles of the ribs and taper off with the pleural reflections at both extremities, where they become quite normal in appearance. Since adjacent ribs frequently are in contact or overlap, synostoses occasionally occur. Likewise, there occur certain concomitant or subsequent alterations of the internal architecture of these deformed ribs.

However, only those ribs and only those portions of the ribs which shelter, or present evidence of having sheltered, empyema cavities show these changes. Evidence of the original extent of empyema cavities has been obtained from roentgenograms taken during the acute stage of the disease and before the establishment of drainage, or from the extent of the thickened pleura as observed in the course of extensive plastic operations or at necropsy.

Some of the material from which these observations were made and brief histories of the cases from which some of the specimens were obtained follow.

REPORT OF CASES

CASE 1.—H. E. M., a boy, 15 months of age, was admitted on May 7, 1932, to the University Hospital with a history of cough, fever and increasing respiratory difficulty for three weeks. From the classic physical and roentgenographic findings

From the Department of Surgery, University of Michigan.

1. Hedblom, C. A.: Deformity of the Thorax Secondary to Pleural or Pulmonary Disease, J. A. M. A. **94**:162 (Jan. 18) 1930.

and the presence of thick pus containing pneumococci, type I, aspirated from the left pleural cavity, a diagnosis of postpneumonic empyema was made.

Daily thoracenteses were carried out for one week. Open drainage was then established and the empyema cavity rapidly became obliterated, so that after three weeks there remained only a short residual sinus. However, two months after admission, death resulted from purulent pericarditis following an intercurrent infection of the upper respiratory tract.

At autopsy an investigation of all the ribs of the left half of the thorax was made. New bone had been deposited on the inferior edges and internal surfaces of all ribs with the exception of the upper two and lower three, in which no alteration could be made out on gross examination. The greatest deposits occurred at the angles, while at the extreme ends of the ribs no abnormality was dis-

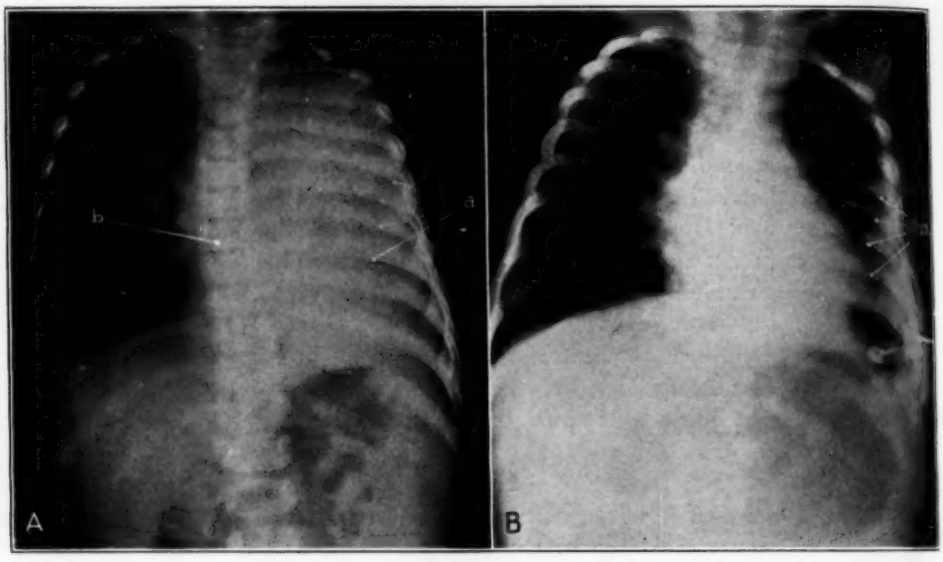

Fig. 1 (case 1).—An undrained acute empyema of less than three weeks' duration: *a*, shadows cast by new-formed bone along the inferior surfaces of ribs 4 to 9 inclusive; *b*, the apex of the functional scoliosis resulting from protective reflex muscle spasm. The narrowing of intercostal spaces frequently observed in chronic empyema is absent. *B*, roentgenogram of the same patient eleven days later and four days after the establishment of drainage. The shadows of new-formed bone (*a*) stand out distinctly against the contrast medium provided by the pneumothorax.

cernible. This is well illustrated in figure 2 *B*, a roentgenogram of the anterior half of one of the ribs. Corresponding to the distribution of new bone deposits, the underlying parietal pleura was firm, dense and thick.

Roentgenograms taken on the patient's admission to the hospital (fig. 1 *A*, before drainage) and a week later (fig. 1 *B*, after drainage) clearly show a layer of new bone along the inferior edge of each rib overlying the empyema cavity. It is important to note that these changes occurred within three weeks after the onset of illness.

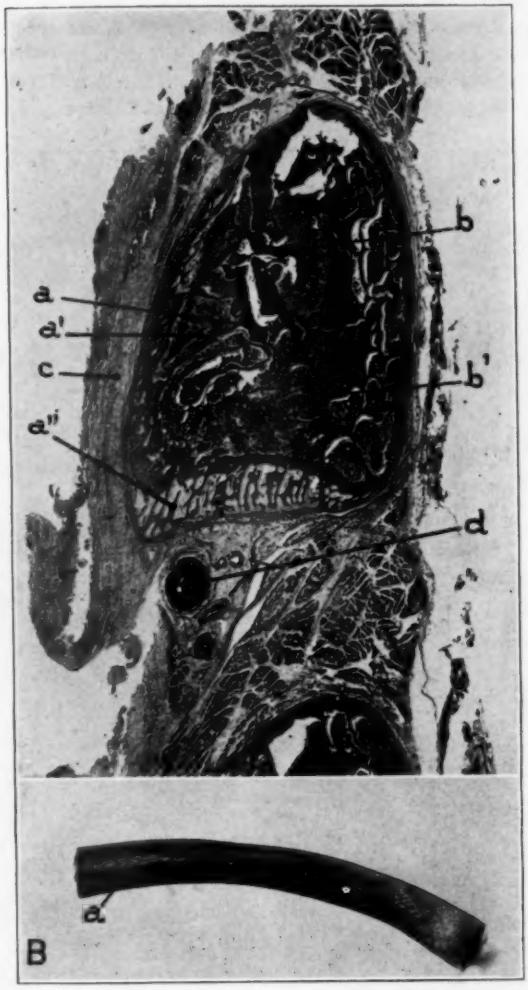

Fig. 2 (case 1).—*A*, upper cross-section of a triangularly shaped rib which overlaid the empyema cavity shown in figure 1. The three strata of new-formed bone (both spongy and dense) responsible for the alteration of the shape of the rib are designated by *a*, *a'*, *a''*; the original internal surface which is buried by new bone and undergoing absorption at *b'* is designated by *b*, the inflamed and greatly thickened parietal pleura and periosteum by *c*, and the intercostal nerve and blood vessels by *d*. Note that the new bone has been deposited on the surfaces adjacent to the inflamed parietal pleura and intercostal blood vessels and that the rib was originally oval in contour. *B*, roentgenogram of the anterior half of the rib illustrated in cross-section in *A*. The newly deposited bone (*a*) diminishes in amount progressively toward the sternal extremity, where for a short distance there is no new bone. On the external surface the cortical thickening from peripheral condensation of trabeculae is apparent.

Two adjoining ribs with their intercostal tissues, periosteum and parietal pleura were cross-sectioned *en masse*. A photomicrograph of the specimen (fig. 2*A*) demonstrates the intimate relation of the new-formed bone to the thickened parietal pleura and the intercostal vessels and its limitation to the surfaces in contact with these structures. It is apparent that this new-formed bone has been deposited in three distinct layers on the old cortex which, although still

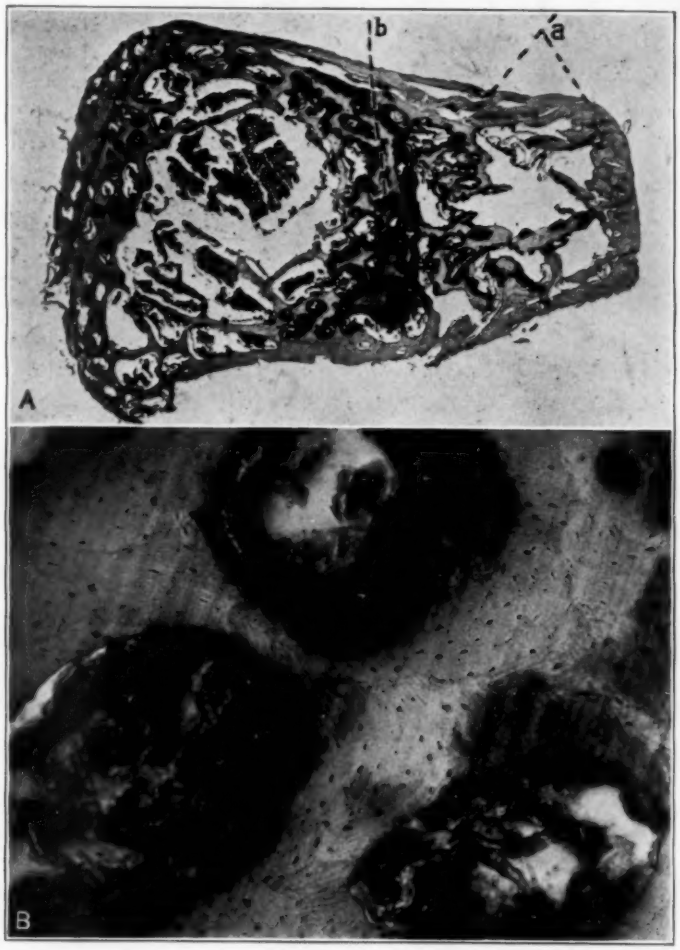

Fig. 3 (case 2).—*A*, photomicrograph of one of the rectangular transformed ribs removed at operation. The old cortex (*b*) which originally was in contact with the inflamed parietal pleura but which is now buried by newly deposited bone (*a*) is surrounded by calcified fibrous marrow. *B*, high power photomicrograph of the calcified fibrous bone marrow illustrated in *A*.

intact, is undergoing absorption, especially at the inferior angle. As a consequence of this transformation the rib has lost its normal oval contour and has become larger and triangular-shaped. The significance of these observations will be considered in the comment.

CASE 2.—C. B., a boy of 15 years was admitted to the University Hospital on July 8, 1929, for treatment of a postpneumonic empyema of the right pleural cavity. The onset of pneumonia had occurred seven weeks previously. Drainage was established immediately on admission, and four months later a Schede thoracoplasty was done to obliterate a large residual cavity with its bronchopleural fistula.

Figure 3 *A*, a low power photomicrograph of a cross-section of one of the ribs, depicts the extensive alteration of external conformation. The new bone, as noted in the preceding case, has been deposited on the cortical surface adjacent to the pleura and in this instance has given the rib a relatively rectangular shape.

Of great interest is the presence of calcified fibrous marrow localized principally about that portion of the old cortex which is enclosed within the newly deposited bone. These changes are represented in greater detail in figure 3 *B* and their possible significance will be considered in the comment.

Fig. 4 (case 3).—From the deposition of new bone (*a*) on the inferior mesial aspect, the contour of the rib, normally oval, has become triangular. Throughout, the bone is extremely dense, probably the result of chronicity of the empyema in a child. The portion of the original cortex buried by the abnormal bone deposit has been absorbed.

The roentgenogram, figure 9 *B*, clearly reveals that the greatest new bone deposits have occurred at the angle, and that the old bone contains an increased number of trabeculae, with definite condensation of these trabeculae about the periphery, especially along the external cortical surface.

CASE 3.—N. S., a boy, aged 5 years, was admitted to the University Hospital on March 5, 1929, with a postpneumonic empyema of the left pleural cavity, which subsequently was proved by biopsy to be tuberculous. A discharging sinus had been present continuously since the establishment of drainage soon after the onset of the disease, two years and three months before admission.

To obliterate a large empyema cavity and to close an associated bronchial fistula, a Schede thoracoplasty was done in stages in January, 1930.

One of the ribs removed at operation (approximately three years after the onset of the empyema) was sectioned for microscopic study. As illustrated in figure 4, extensive transformation of the rib has occurred. In cross-section it is considerably enlarged and definitely triangular. The cortex is abnormally thick

and composed of abnormally dense bone. As has occurred in all cases which have been studied, the new bone has been deposited on the surface of the rib adjacent to the pleura and the intercostal blood vessels. The portion of the original cortex on which the new bone was laid down has been entirely absorbed.

CASE 4.—A. W., a white man, aged 22 years, entered the University Hospital on Dec. 8, 1929, for the treatment of a chronic, postpneumonic nontuberculous empyema of ten months' duration. Soon after the onset of empyema, intercostal drainage had been established, but one month later the sinus had been permitted to close. Subsequently spontaneous drainage through a bronchus had occurred.

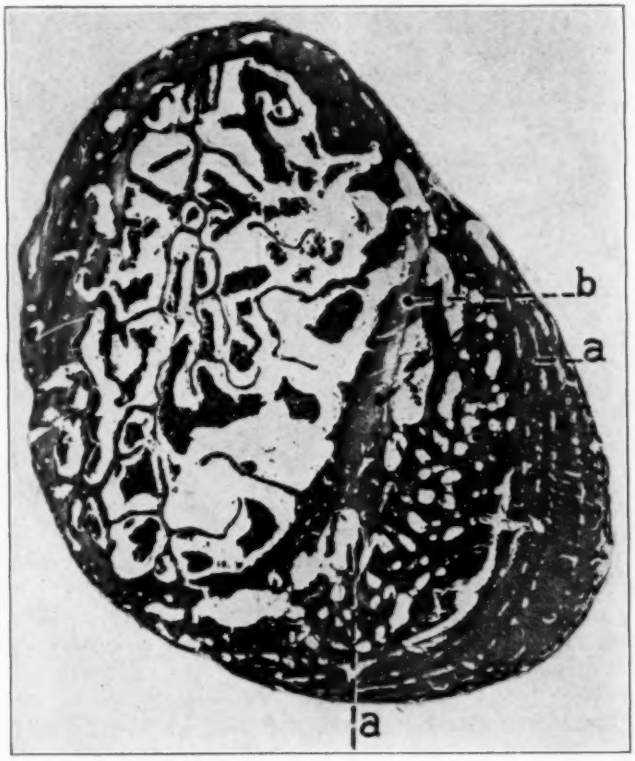

Fig. 5 (case 4).—The outline of the original cortex of the transformed rib is definitely oval. The new bone (*a*) has been laid down in several layers under that portion of the periosteum which is in contact with the intercostal blood vessels and inflamed parietal pleura. The new bone is rather dense and the marrow adjacent to the buried old cortex (*b*) presented in several sections fibrous tissue replacement of the normal marrow tissue.

On the day following admission, adequate dependent open drainage was established and prompt obliteration of the cavity ensued.

The alterations of external conformation and of the internal architecture of the rib resected approximately five months after the onset of the empyema are depicted in figures 5 and 9 *C*. The new bone has been laid down on the inner

surface and inferior border of the rib in semicompact concentric lamellae. The portion of the old cortex on which the new bone has been deposited has remained intact. In some marrow spaces of the old bone there has been replacement of normal marrow by fibrous tissue.

The roentgenogram of this rib (fig. 9*C*) brings out the dense newly deposited bone in sharp contrast to the less dense old bone. The other evidences of transformation noted in the previously described specimens are present but to a lesser degree.

EXPERIMENTAL OBSERVATIONS

EXPERIMENT 1.—Dog 78F, an adult mongrel police dog, weighing 14 Kg., was operated on. After a subperiosteal resection of a 3.5 cm. segment of the right

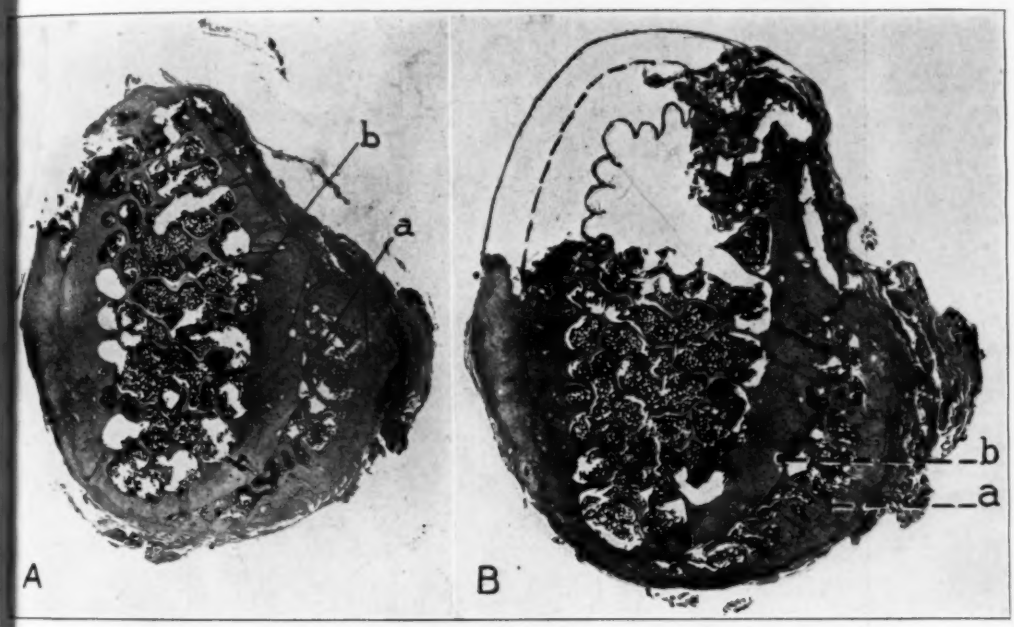

Fig. 6.—Ribs from experimental animal in experiments 1 (*A*) and 2 (*B*). Typical subperiosteal new bone (*a*) has been laid down on the cortex (*b*) under that portion of the periosteum which normally is in contact with the parietal pleura. The gross architecture of the old rib has been reproduced. Compare these specimens with the human specimen in figure 2*A*.

tenth rib at its angle, the endothoracic cleavage plane was established and an extrapleural pneumolysis was performed; the parietal pleura was gently freed from the periosteum of ribs 9, 8 and 7 and their respective intercostal structures for a distance of approximately 10 cm. Into the space created by the pneumolysis, gauze was gently packed and the wound closed without drainage.

Healing occurred by primary intention, but on the third postoperative day and on four subsequent occasions it was necessary to aspirate considerable serosanguineous fluid from the subcutaneous and deeper tissues.

Six weeks after operation the animal was killed and at autopsy there was a small subcutaneous collection of serous fluid containing flecks of fibrin and com-

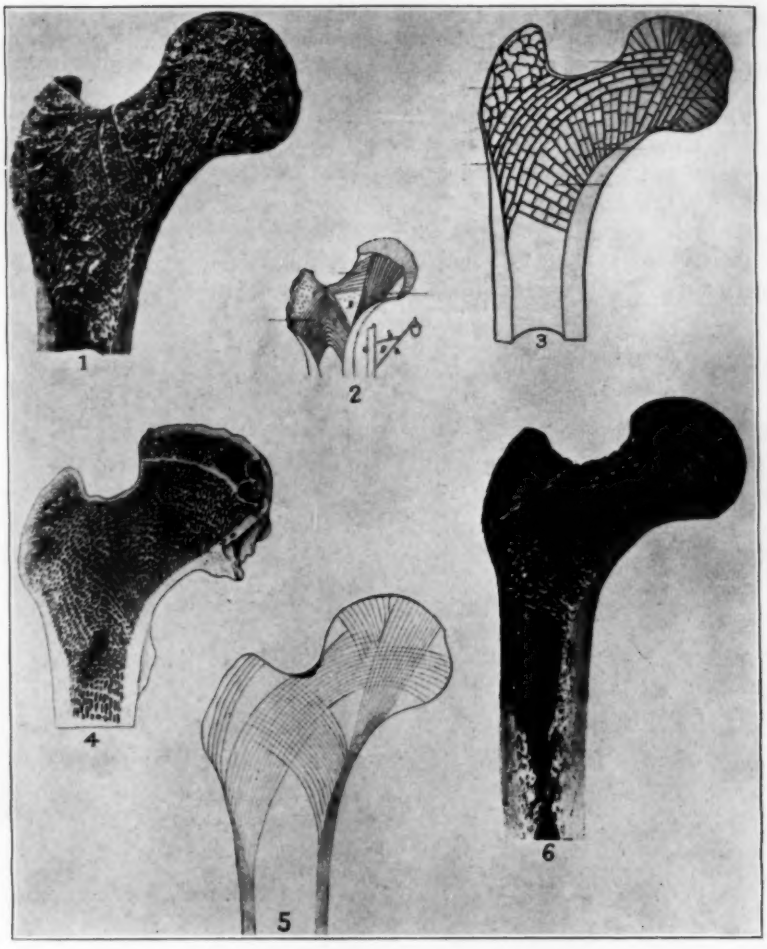

Fig. 7.—Reproduction of an illustration from Wolff (Das Gesetz der Transformation der Knochen, Berlin, A. Hirschwald, 1892): Nos. *1* and *6*, photographs of the bony architecture of the proximal portion of the shaft and of the head and neck of the normal femur. No. *2*, this crude but effective analogy of the mechanical support given by trabeculae to the overhanging head and neck on the shaft of the femur for the transmission of the forces of weight-bearing to the mechanics of the lamp-post bracket was first used by Ward, an English anatomist. Nos. *3* and *4*, diagrammatic representations of the inner or trabecular architecture, demonstrating the two principal systems of trabeculae; the compression system arising from the adductor side of the cortex of the shaft and extending in a fan-shaped arrangement to the greater trochanter and the opposite surfaces of the head and neck, and the traction or tensile system which arises from the cortex of the abductor side of the shaft and extends in a wide arch to the adductor portion of the head. These systems are supplemented by smaller groups; they always course either perpendicularly or parallel to the axis of the bone, and intersect at right angles. No. *5*, diagrammatic illustration of the courses of the trabecular systems described.

municating with the subcostal space. The gauze pack was rather firmly fixed to the greatly thickened pleura and to the chest wall by a fibrinous exudate and some granulation tissue. The inner, or that portion of the periosteum in contact with the gauze was greatly thickened, edematous and vascular. It separated from the ribs with abnormal ease. The normal oval contour of each rib had been altered by a layer of new bone on its internal surface, the extent corresponding exactly to that of the inflamed portion of the periosteum with which it was in contact.

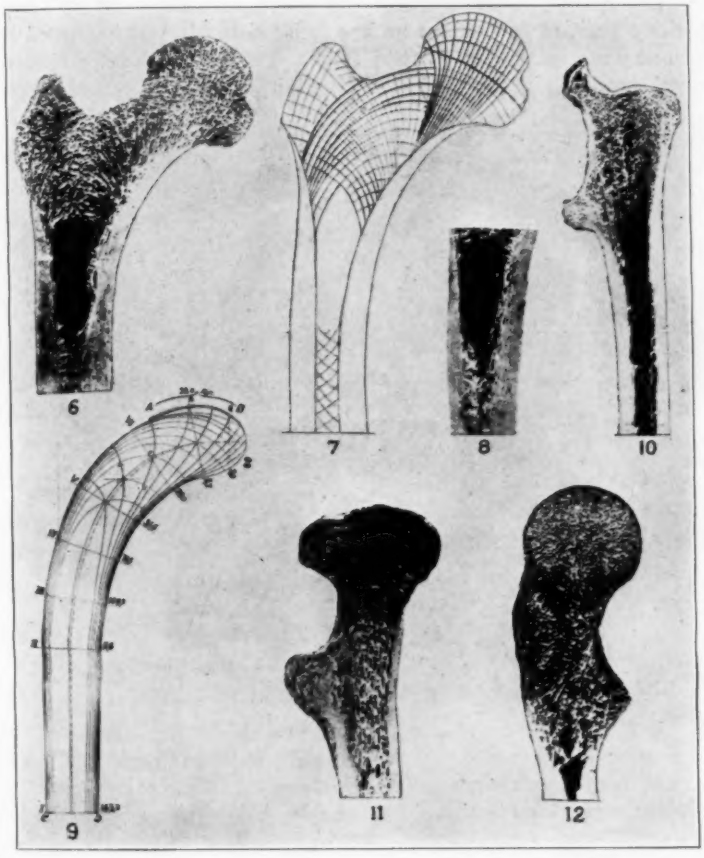

Fig. 8.—Reproduction of an illustration taken from Wolff (Das Gesetz der Transformation der Knochen): Nos. 6, 8, 10, 11 and 12. Sections of the femoral head, neck and shaft which were cut in various planes for a complete analysis and reconstruction of the trabecular systems. The mathematical basis of Wolff's law is depicted in the striking similarity of the diagrammatic representation of the trabecular systems (*7*) to the graphic representation of the distribution of forces in their various components (mathematically calculated) in a structure, the Fairbairn crane (*9*), which is similar in shape and function to the human femur.

As revealed by microscopic examination (fig. 6*A*) of a cross-section of one of these ribs (the eighth), the transformation had resulted from the deposition of

a crescent of subperiosteal new bone on the old cortex, histologically not unlike that observed in the human specimens (figs. 2*A* and 5), a newly formed subperiosteal cortex of dense bone with a central spongiosa of trabeculae and of fatty and cellular marrow.

EXPERIMENT 2.—Dog 84F, an adult shepherd dog, weighing 12.5 Kg., was operated on. With a technic similar to that of the pectoral muscle pneumolysis described by Alexander,[2] long segments of ribs 7, 8, 9 and 10 were stripped of their periosteal sheaths, and a muscle flap raised from the chest wall and fashioned with a pedicle to preserve an adequate blood supply was insinuated between the internal surfaces of the bare ribs and the mesially displaced periosteum and intercostal bundles. The wound was closed in layers and healed by primary

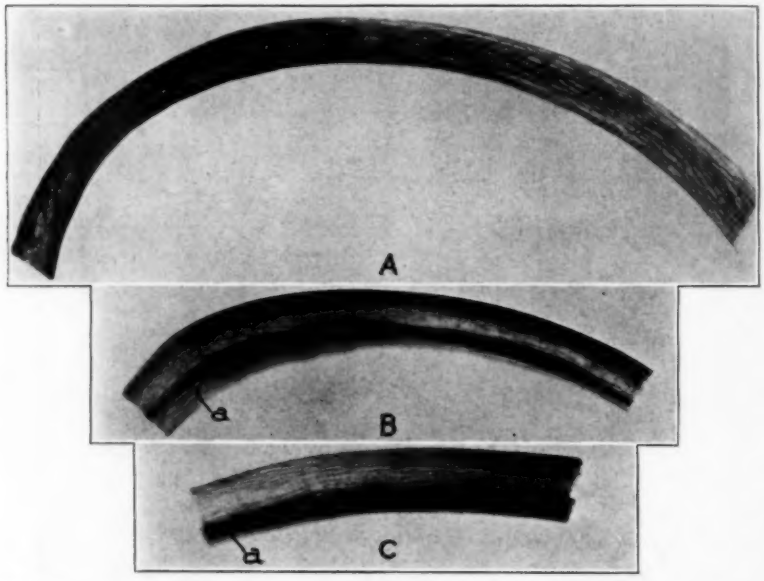

Fig. 9.—*A*, roentgenogram of a normal rib with the flat surface against the film. Posterior to its angle the rib becomes relatively round and its cortex thicker, and its spongiosa contains fewer trabeculae, which cross diagonally from one opposing cortical surface to the other. In the anterior portion the longitudinal trabeculae can be demonstrated traversing the long axis of the rib for long distances, and the cortex is very thin. *B*, roentgenogram of a transformed rib removed in case 2. Note the new-formed bone (*a*) deposited in greatest quantities at the angle of the rib and in progressively smaller quantities anteriorly. In comparison with the normal rib structure in *A*, the cortex is greatly thickened at the expense of the trabeculae of the spongiosa. *C*, roentgenogram of a rib removed in case 4. There is an increase of trabeculae, no thickening of the cortex, but the newly deposited bone (*a*) on the inner surface is very dense. The greatest density and quantity are found at the angle.

2. Alexander, John: Supraperiosteal and Subcostal Pneumonolysis with Filling of Pectoral Muscles, Arch. Surg., to be published.

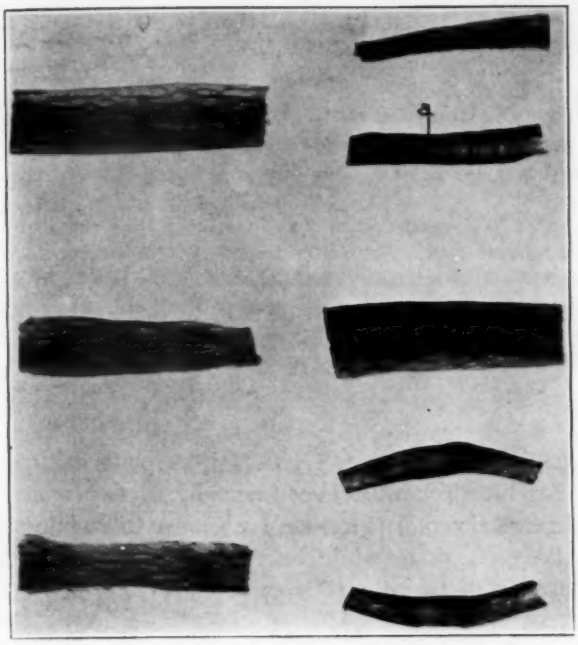

Fig. 10.—Roentgenograms of normal ribs, which demonstrate the two systems of trabeculae: Those of the first are placed longitudinally to the long axis of the rib, and are intersected by those of the second and smaller system, which are transversely situated to the long axis of the rib and which bridge opposing cortical surfaces. Note the thickened cortex in the section cut posterior to the angle of the rib (*a*).

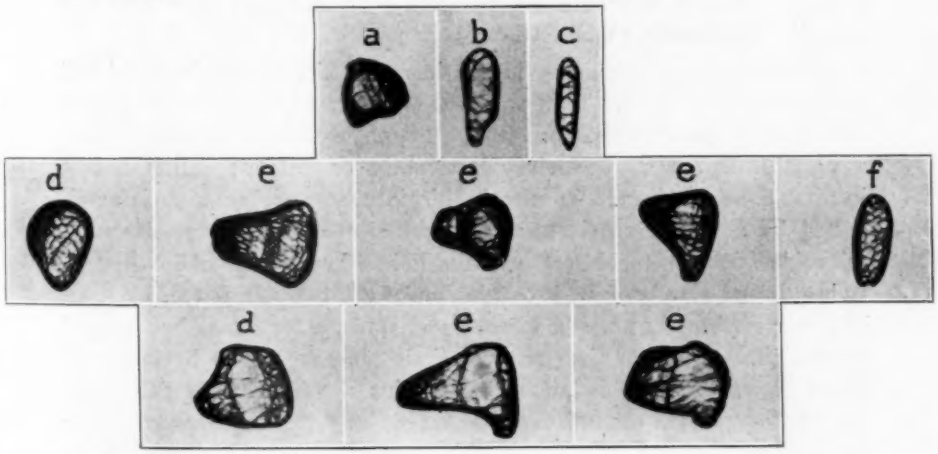

Fig. 11.—Roentgenograms of cross-sections of a normal rib, posterior to the angle of the rib near its vertebral articulation (*a*) at the angle (*b*) and at the sternal end (*c*), and of two transformed ribs posterior to the angle of the rib (*d*), at the angle (*e*) and at the sternal end (*f*). In the latter group note the evidence of greatest deposits of new bone at the angles and progressively diminishing toward both extremities, the absence of new bone at the sternal end and consequently a normal oval contour, the thickened cortex and the increase in trabeculae throughout. The cortex of the section of the normal rib (*a*) is thicker than that of the transformed ribs (*d*) because it was cut at a greater distance posterior to the rib angle and nearer its vertebral articulation.

intention. On the eighth postoperative day considerable serosanguineous fluid was aspirated from the subcutaneous tissues. Cultures of it were sterile.

The animal was killed sixty days after operation. The rib segments which had been denuded of periosteum were partially necrotic, and the portion of the mesially displaced periosteum which was in contact with the interposed muscle had produced no new bone. But at the borders of the interposed muscle where the periosteum was in direct contact with neither muscle nor the cortex of the rib from which it had been lifted, a layer of new bone had been deposited on the rib, between its cortex and the periosteum. A cross-section of one of these ribs at this site (fig. 6*B*) presents a picture almost identical with that observed in experiment 1 (fig. 6*A*). Consequently its histologic changes need no further comment.

It is apparent that the same physiologic response of the rib and its periosteum has been produced experimentally by two widely divergent methods. In experiment 1 there was a response to aseptic inflammation of foreign body reaction and in experiment 2 a response to traction on and separation of periosteum from bone.

COMMENT

In an endeavor to correlate these clinical and experimental observations and to explain the cause and nature of the transformation observed in these ribs two physiologic processes or principles will be considered. These may be designated as the osseous and periosteal response to (1) inflammation, periostitis or osteoperiostitis and (2) functional demands (Wolff's law [3]).

Inflammatory Response.—The intimate relation of the inflamed and infected parietal pleura to the adjacent surfaces of the overlying ribs and periosteum with their respective lymphatic communications makes a certain degree of secondary periostitis or osteoperiostitis almost inevitable. As observed in case 1, this inflammatory reaction is manifested by a marked periosteal and subperiosteal cellular proliferation (fibroblasts and osteoblasts) and the deposition on the old cortex of trabeculae of new bone with cellular and fatty marrow spaces enclosed in a newly formed cortex of dense bone. This process may be repeated several times to form tiers of cancellous bone separated by layers of dense bone. From this picture one gains the impression that there have been repeated stimuli to osteogenesis, and to each a reproductive

3. Wolff, J.: Das Gesetz der Transformation der Knochen, Berlin, A. Hirschwald, 1892; Ueber die Wechselbeziehungen zwischen der Form und der Funktion einzelnen Gebilde des Organismus, Leipzig, F. C. W. Vogel, 1901; Die Lehre von der funktionellen Pathogenese der Deformitäten, Arch. f. klin. Chir. **53**:831, 1896; The Law of Transformation of the Internal Architecture of Bones in Pathological Changes of the External Form of the Bone, Sitzungsb. d. K. Pr. Akad. d. Wissensch., Berlin **1**:475, 1884.

response. In figure 2 *A*, there may be discerned three separate strata or attempts at new rib formation.

This same architectural arrangement of spongy and cortical new bone and its deposition in layers is observed frequently in subperiosteal new bone formation in long bones (Kauffmann [4]). Likewise an analogous condition occurs not infrequently in an extremity in which there is a cellulitis with the infection superficial to the periosteum. The periosteum takes part in the regional inflammatory reaction, and the ensuing osteogenic response produces a histologic picture of newly deposited bone precisely the same as that observed in the rib specimens which were in contact with the infected parietal pleura. I recently studied two such specimens, both obtained from the external surface of the tibia.

The significance of fibrous replacement of the normal cellular and fatty marrow of the old bone of two of the rib specimens (figs. 3 and 5) and of its calcification in the one instance (fig. 3 *B*) is somewhat problematic. Since these pathologic changes elsewhere in the body, excepting metabolic disturbances and new growths, occur in tissues of low vitality which have been the seat of chronic low grade infection or in tissues which have undergone necrosis, it is reasonable to interpret them as evidence of degeneration. Brunschwig [5] has reported the occurrence of calcified fibrous bone marrow in three cases of chronic osteomyelitis of over fifty years' duration. In two of the cases it occurred only in dead bone which had been sequestrated or which was undergoing sequestration, but in the third case there was calcification in fibrous marrow of living bone some little distance from a large osteomyelitic cavity. He [6] also reported the observation of extensive calcification in the dense fibrous lining of an old bone cyst. Phemister [7] has noted its occurrence in tuberculous sequestrums. It is of interest that Brunschwig, in an extensive search of the literature, could find no record of similar observations.

To make deductions by analogy from this small amount of data would be absurd. However, the possibility of preexistent low grade nonsuppurating osteomyelitis of these two ribs with subsequent fibrosis and calcification is suggested. That these changes occurred principally in the marrow nearest the infected empyema cavity may have similar significance.

4. Reimann, in Kauffmann: Pathology for Students and Practitioners, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 2.

5. Brunschwig, A.: Epithelization of Bone Cavities and Calcification of Fibrous Marrow in Chronic Pyogenic Osteomyelitis, Surg., Gynec. & Obst. **52:** 759 (March) 1931.

6. Brunschwig, A.: Solitary Bone Cysts of Long Duration, J. Bone & Joint Surg. **12:**141, 1930.

7. Phemister, D. B.: Necrotic Bone and the Subsequent Changes Which It Undergoes, J. A. M. A. **64:**211 (Jan. 16) 1915.

In experiment 1 an aseptic inflammatory reaction was produced in that portion of the rib and its periosteum which normally is in contact with the parietal pleura. Since this stimulation of osteogenesis produced results almost identical to those observed in the human ribs described, it may be deduced that both represent the normal physiologic response of bone and periosteum to inflammation.

To these facts which have been cited in favor of the "response to inflammation" may be added the observation made in case 1. In less than three weeks after the onset of empyema and before the establishment of drainage there was roentgenographic evidence of new bone formation on the inferior borders of the ribs overlying the empyema cavity. It seems unlikely that under these circumstances and at such an early stage of the disease there would be any contracture of pleural scar or sufficient alteration of the forces to which the ribs are subjected to bring about a transformation of architecture.

As previously pointed out, the deposition of new bone in all the specimens was limited to that portion of the ribs which was adjacent to the inflamed parietal pleura and to the intercostal vessels.

Functional Response.—In 1872 Julius Wolff [3] stated his law of transformation of bone which revolutionized many of the contemporary concepts of bone deformities. It may be interpreted as follows: Every change in the form and function of bones or of their function alone is followed by certain definite changes in their internal architecture and equally definite secondary alterations of their external conformation in accordance with certain mathematical laws. This law was the culmination of a long painstaking study of the architecture of various bones of the body under normal and abnormal conditions. It embodied the observations recorded by many investigators dating from Galileo, and the mechanical principles formulated by Cullmann [8] and elaborated on later by Meyer.[9] These principles are illustrated in figures 7 and 8, reproductions of illustrations from Wolff's monograph. This fascinating bit of medical history has been reviewed in detail by Koch,[10] Keith,[11] Monro,[12] Albert [13] and others, and from their manuscripts have been

8. Cullmann, quoted by Wolff (footnote 3) and Keith, A.: Menders of the Maimed, London, Oxford University Press, 1919, p. 277; footnote 11.

9. Meyer, G. H.: Die Statik und Mechanik des menschlichen Knochengerüstes, Leipzig, W. Engelmann, 1873.

10. Koch, J. C.: The Laws of Bone Architecture, Am. J. Anat. **21**:177, 1917.

11. Keith, A.: Hunterian Lecture on Wolff's Law of Bone Transformation, Lancet **1**:250, 1918.

12. Monro, A.: A System of Anatomy and Physiology, Edinburgh, Creech, 1795.

13. Albert, E.: Einfuehrung in das Studium der Architektur der Roehrenknochen, Vienna, A. Hölder, 1900.

taken excerpts which may assist in a clearer definition of Wolff's law in respect to its application to the problem under discussion.

Monro [12] credits Galileo (1638) with the earliest mention of the mechanical importance of the architecture of bone and its relation to his discoveries in the applied mechanics of beams. Although numerous subsequent observers contributed anatomic descriptions of the inner architecture of bone (Duhamel [14] in 1743, Havers,[15] Loder [16] in 1805 and Bourgery [17] in 1832), the next evidence of an appreciation of its mechanical and functional significance was recorded in 1838 by Ward,[18] an English anatomist. His studies formed the anatomic basis of Wolff's law.

The second basic principle of the law came through the recognition that bone is not a fixed tissue, but is continually in a state of flux. In regard to this phenomenon Hunter [19] observed that bone "is constantly changing its matter," and Flourens,[20] in 1842, stated that there is a continual mutation of matter, old molecules being absorbed and new ones laid down. In discussing this inherent ability of bone to reconstruct itself, Keith [21] has stressed the significant rôle played by osteoblasts, "the architects and hod-carriers" of bone. He draws an analogy to the scleroblasts of marine life and refers to Woodland's [22] investigations of spicule formation in sponges. To resist the forces of moving water, the soft tissues of sponges are supported by a calcareous skeleton deposited by cells designated as scleroblasts. These cells are found in great numbers arrayed in a fashion about new forming spicules similar to that of osteoblasts about trabeculae in the process of deposition. Apparently they are highly sensitive to vibrations and stresses, and should the position of the sponge be altered in relation to the forces playing on it, such as a reversal of the surfaces exposed to the forces of the waves, the trabecular framework, through the action of scleroblasts, undergoes extensive reconstruction and is fashioned to resist the forces to which it is then subjected.

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14. Duhamel, quoted by Koch.[10]

15. Havers, quoted by Keith.[11]

16. Loder: Tabulae anatomicae vimariae, 1805; quoted by Koch.[10]

17. Bourgery, J.: Traité complet de l'anatomie de l'homme, Paris, C. A. Delaunay, 1832.

18. Ward, F. O.: Outlines of Human Osteology, London, H. Renshaw, 1838.

19. Hunter, J.: Complete Works, London, Longman, 1837, vol. 2.

20. Flourens, J. P.: Recherches sur le développement des os et des dents, Paris, Gide, 1842.

21. Keith, A.: Bone Growth and Bone Repair, Brit. J. Surg. **6**: 160, 1918-1919.

22. Woodland, W.: Studies in Spicule Formation, Quart. J. Micr. Sc. **69**: 231, 1906.

From the observations of Cullmann,[8] Wolff obtained the third principle involved in his law. While observing an anatomic exhibit prepared by von Meyer, Cullmann, a Viennese mathematician, was impressed with the remarkable resemblance existing between the internal bony architecture of the head, neck and shaft of the normal human femur and the graphostatic diagram of a Fairbairn crane. By applying a load of 30 Kg. (the approximate load borne by an adult human femur) to the crane and by projecting the resultant lines of force, he was able to demonstrate a definite analogy in the courses of the trabeculae of the head and neck of the femur as seen in the frontal section and in those of the trajectories of his diagram. He concluded that bone was constructed in accord with mechanical principles to provide the greatest strength and efficiency with a minimum of material.

In a subsequent investigation of a large number of bones which were deformed as a result of abnormal function, trauma and disease, Wolff [23] demonstrated the observance of the same mathematical principles in these reconstructed and transformed bones. Since any change in the external form of a bone is followed by a change in the direction of the forces of traction and pressure acting on it, a transformation of its inner architecture along these lines of force should occur in maintenance of mechanical efficiency. That this does occur, and according to mathematical principles, Wolff demonstrated by determining the changes mathematically before verifying them anatomically. Apparently trabeculae which have been rendered statically useless by a change in the form of the bones are destroyed and are replaced by new trabeculae adjusted to the changed form.

From a very laborious study Koch [10] calculated the forces of weight-bearing in all their components acting on each system of trabeculae of the femur at various levels, and determined the relative factors of safety at these levels. From these calculations he was able to predict with a high degree of accuracy the relative frequency of fractures at various levels when forces acting along their normal axes were increased beyond the factor of safety.

Gallois and Japiot [24] studied the inner bony structure of vertebrae (predominantly spongy bone) and arrived at conclusions confirmatory of Wolff's law. From a roentgenographic study of femora of various species of animals, Freiberg [25] demonstrated a variance of architecture consistent with the variation in function.

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23. Wolff, J.: Bemerkungen zur Demonstration von Roentgenbildern der Knochenarchitectur, Berl. klin. Wchnschr. **37**:381, 1900.

24. Gallois and Japiot: Architecture intérieure des vertèbres, Rev. de chir. **63**:688, 1915.

25. Freiberg, A. H.: Wolff's Law and the Functional Pathogenesis of Deformity, Am. J. M. Sc. **124**:956, 1902.

It is reasonable to assume that the bony architecture of the ribs has been constructed on the same mechanical principles as all other bones, and that every change in the forms and functions or functions alone would result in changes in internal architecture and external conformation. The complexity of the functions for which ribs are designed makes an analysis of the forces acting on them and a projection of those forces impossible. The stresses of superincumbent weight have little importance compared to the muscle pull of respiratory excursions and the stress incident to the unequal pressures on the internal and external surfaces of the ribs during the inspiratory phase of respiration. Likewise, these forces are distributed over a large area and are shared to a small extent by other structures such as the vertebral column. Unquestionably, the bony thoracic cage possesses a large factor of safety for the protection of the enclosed viscera against external violence. Equally uncertain are the appraisal and analysis of the alteration of forces of stress, compression and torsion resulting from a chronic infection of the pleural cavity. In the acute stage of the disease the affected side usually is splinted by spasm of the muscles not only of the hemithorax but also of the abdomen, back and neck of the same side. With progression to chronicity and in the process of healing the pleural scar contracts and exerts increased and abnormal forces on the overlying ribs.

In accord with Wolff's law a transformation of the costal bony architecture to meet these abnormal forces would be expected, and the transformed ribs would represent functional rather than pathologic deformities. In an endeavor to investigate the changes of the inner architecture of these abnormal ribs as compared to the normal, microscopic and roentgenographic studies of sections cut in transverse, longitudinal and sagittal planes were made. As illustrated in figures 9 *A* and 10 the cancellous structure of the normal rib consists of two principal systems of trabeculae. The larger and thicker trabeculae traverse for long distances the long axis of the rib. They leave the cortex at rather acute angles, and at the angle of the rib they appear to cross the cancellous space and join the opposite cortex (probably to buttress this segment which receives greatest stress). The second or smaller system is composed of short trabeculae placed roughly at right angles to the long axis of the rib. Since these trabeculae irregularly intersect those of the longitudinal system to produce a honey-comb structure, it is impossible definitely to trace their courses, but it would appear that they tend to bridge opposing cortical surfaces. Posterior to its angle each rib, with the exception of the first, becomes somewhat more round in contour. The cortex is much thicker and the spongiosa contains comparatively few trabeculae. This fact and the stratified composition of the cortex of some ribs (fig. 4) add support to the

contention of von Meyer and Wolff that the cortex of all bones is made up from a condensation of the peripheral portions of the trabeculae of the spongiosa.

The inner architecture of the abnormally shaped ribs presents definite changes. The longitudinal trabeculae are increased in numbers and show marked peripheral condensation to form a relatively thick but loosely packed cortex (figs. 2 *B* and 9 *B*).

The occurrence and significance of fibrous replacement of normal marrow and of calcified fibrous marrow have been discussed. Similarly the subperiosteal deposits of new bone have been dealt with. To explain the changes in the marrow on the basis of a purely functional response is impossible. The subperiosteal new bone may in part represent a transformation in accordance with Wolff's law. Although much evidence, already enumerated, indicates that the periostitis or inflammatory response initiates osteogenesis, subsequent stimuli may come from contraction of the pleural scar.

Experiment 2 was designed to determine this response of periosteum and bone to tension on the periosteum and its separation from the bone. New bone was laid down in precisely the same manner as occurred in response to inflammation in experiment 1 and as has been observed in the human specimens. From the results of this experiment it may be deduced that tension from contraction of the pleural scar of chronic empyema may initiate secondary stimuli to osteogenesis and deposition of the younger tiers of subperiosteal new bone, as illustrated in case 1 (fig. 2*A*).

SUMMARY AND CONCLUSIONS

1. The structural changes commonly observed in ribs which overlie empyema cavities and the relation of these changes to the parietal pleura are described.

2. A report of an investigation and certain deductions relative to the pathogenesis of these changes are presented. This analysis, based on clinical, histologic, roentgenographic and experimental data, indicates that the transformation has been brought about by the action of two physiologic processes.

The first of these, the inflammatory response or periostitis which probably is most active during the acute stage of an empyema, occurs in that portion of the rib and its periosteum which is in contact with the dilated intercostal vessels and the inflamed and infected parietal pleura and causes the laying down of one or more distinct layers of subperiosteal new bone. Further evidence of a secondary inflammatory response and possibly of infection may be deduced from the presence in two rib

specimens of fibrous and calcified fibrous bone marrow. These degenerative changes suggest the possibility of a preexistent osteitis or osteomyelitis.

The second factor has been designated as a functional response in which the osteogenic elements of these ribs bring about certain transformations in accord with the principles outlined in Wolff's law. It is probable that the proliferation of trabeculae and their peripheral condensation to form the thickened cortex observed in the transformed ribs represent a functional response to certain abnormal forces acting on the ribs as a result of contracture of the pleural scar of the underlying empyema. Likewise, this same physiologic process may be in part responsible for the subperiosteal deposits of new bone.

It would appear that the greatest abnormal force acting on these ribs overlying chronic empyema cavities is the centripetal traction of the contracting pleural scar. To resist this force the transformation of the inner architecture constantly observed in the specimens examined would logically be anticipated.

Dr. John Alexander permitted the use of his collection of pathologic ribs in this study. Dr. C. V. Weller and Dr. J. C. Bugher assisted in the preparation of the pathologic specimens.

COMBINATION AVERTIN-ETHER RECTAL ANESTHESIA

EXPERIMENTS ON ANIMALS

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The purpose of the experiments here described was twofold: first, to determine whether a combination of avertin (tri-bromethanol) and oil-ether, administered by rectum in experiments on animals, would produce anesthesia measurably deeper or more lasting than avertin alone; and second, to determine the relative safety of these drugs when thus used. The work was undertaken in the hope of securing a combination of anesthetic effects which would give a safe anesthesia in man with the comfortable, rapid induction characteristic of avertin and the more sustained, deeper anesthesia induced by ether, making supplementary inhalation anesthesia unnecessary for operations for which marked relaxation is not required.

LITERATURE

The development of tri-bromethanol in Germany and its introduction into this country some years ago have given rise to an extensive literature, originally in German, but more recently in English also. This literature has been well summarized by many American writers, notably by Waters and Muehlberger,[1] who in addition reported experimental and clinical investigations. However, in only one article, by York and Schork,[2] have I found any reference to the use of rectal ether as a supplement to avertin anesthesia. They reported the use of this combination for several patients, with excellent results, but had not done any experimentation on animals with the procedure.

EXPERIMENTS WITH ALBINO RATS

The first experiments were conducted with albino rats, which have the double advantage of being easy to handle and of having relatively capacious rectums. The technic used was as follows:

No preliminary medication was given. A small amount (from 10 to 20 cc.) of 3 per cent aqueous solution of avertin was prepared just before each group of

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From the Department of Surgery, College of Physicians and Surgeons, Columbia University. This experimental work was done while the author was a Junior Fellow in Surgery.

1. Waters, Ralph M., and Muehlberger, C. W.: Tribromethanol Anesthesia. Arch. Surg. **21**:887 (Dec.) 1930.

2. York, Arthur J., and Schork, R. J.: Tribromethanol as a Basal Anesthetic, South. M. J. **24**:244, 1931.

experiments and kept at 40 C. over a water bath. For each animal the dose of avertin was calculated from the body weight, and the corresponding quantity of 3 per cent avertin solution was measured into a syringe and injected into the rectum through a 2½ to 3 inch segment of a small rubber catheter. Care was used to have the lumen of the catheter filled with solution, so that all of the

TABLE 1.—*Experiments on Albino Rats with Avertin Alone, with Avertin-Ether and with Ether Alone*

Avertin in Gm. per Kg.	Ether in Cc. per Kg.	Number of Rats	Length and Depth of Anesthesia and Result
0.15	..	2	Drowsiness only; recovery for both
0.3	..	7	1 rat, complete anesthesia for 1½ hours, recovery 6 rats, drowsiness and partial anesthesia, all recovered
0.5	..	2	1 rat, anesthesia for 3 hours, recovery 1 rat, anesthesia for 5 hours, recovery
0.6	..	2	1 rat, anesthesia for 1 hour, recovery 1 rat, anesthesia for 3 hours, recovery
0.7	..	2	1 rat, death after ½ hour 1 rat, anesthesia for 2 hours, recovery
0.3	1	4	1 rat, death after 1½ hours 1 rat, partial anesthesia for 1 hour 1 rat, complete anesthesia for 1 hour 1 rat, complete anesthesia for 2 hours
0.3	2	5	1 rat, death after 20 minutes 1 rat, anesthesia for 1 hour 1 rat, anesthesia for 1½ hours 2 rats, anesthesia for 2½ hours
0.3	4	5	1 rat, death after 50 minutes 1 rat, anesthesia for 2 hours 3 rats, anesthesia for 3 hours
0.3	5	6	5 rats, in 2, death after 4½ or 5 hours; 3 found dead in the morning; in 3 of the 5 signs of recovery at one time or another 1 rat, anesthesia for 4½ hours, recovery
0.3	6	1	1 rat, death after 2 hours
0.4	5	2	2 rats, death; one after 10 minutes; one after 3 hours
0.4	10	2	2 rats, death after 3 or 3½ hours
...	2	1	1 rat, partial anesthesia
...	4	3	3 rats, partial anesthesia
...	8	2	1 rat, death during night 1 rat, partial anesthesia only
...	10	4	1 rat, anesthesia for ½ hour 3 rats, partial anesthesia only
...	15	2	1 rat, death during night 1 rat, partial anesthesia only

measured quantity was injected. After several methods of holding the rats had been tried, best results were obtained by tying them in the dorsal position to a small animal board. During and after injection, compression of the preanal region against the extended tail prevented escape of the injected solution. The rats were deemed anesthetized when they fell to the cage floor and lay motionless, and anesthesia was assumed to continue until they first made ataxic efforts to regain their feet, or until they responded to painful stimuli, such as application of a Kocher clamp to the skin.

In the experiments in which ether was used, a solution composed of two parts of ether and one part of liquid albolene was made, and the doses were calculated, measured and injected in the manner described. When the avertin-ether combination was used, the avertin was injected first, and the oil-ether from five to ten minutes later, after the animal was anesthetized.

A complete tabulation of these experiments will be found in table 1.

These experiments were not expected to give conclusive proof of either the efficacy or the safety of avertin-ether anesthesia, but were intended merely as preliminary guides in undertaking the more extensive and accurate work on dogs. There was conclusive evidence, however, that doses of avertin and ether which separately induced only partial anesthesia, when given together produced a deep anesthesia lasting from one to three hours; for example, avertin, 0.3 Gm. per kilogram, plus ether, 4 cc. per kilogram.

Because of the mortality, from 20 to 25 per cent, with the smaller doses of this combination, the work with dogs was approached with caution.

EXPERIMENTS WITH DOGS

This work involved a good deal of trial and error. In the first twenty-three experiments, the dogs were given no preliminary medication. With several dogs, enemas were used as preanesthetic preparation; others were starved for twenty-four hours; in a third group both methods of preparation were used; in a fourth group, neither.

In these experiments, avertin alone in doses of from 0.1 to 0.3 Gm. per kilogram gave anesthesia lasting from one-half to one hour. Addition of ether in doses of from 1 to 2 cc. per kilogram slightly increased the length of anesthesia though the maximum duration obtained was one hour and twenty minutes (with avertin, 0.3 Gm. per kilogram, plus ether, 2 cc. per kilogram); but the majority of these experiments were unsuccessful because of expulsion of the oil-ether mixture either immediately or after some minutes. In an effort to overcome this difficulty, ether was emulsified with milk and injected; and in another case a solution was made by dissolving the calculated amount of avertin in the calculated amount of oil-ether mixture. Both of these procedures failed; the ether-milk mixture was expelled as promptly as the oil-ether, and the avertin-oil-ether solution produced only drowsiness.

At Dr. Gwathmey's [3] suggestion, a preliminary medication of morphine, 10 mg. per kilogram, was then instituted, as recommended by Beckman.[4]

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3. Gwathmey, James T.: Personal communication.

4. Beckman, Harry: On the Alleged Synergism of Magnesium Sulfate and Morphin When Injected Prior to the Induction of Anesthesia by the Oil-Ether Colonic Method, J. Lab. & Clin. Med. **10**:189, 1924; Colonic Anesthesia in Dogs, J. Am. Vet. M. A. **20**:67, 1925. (This gives a somewhat more complete description of the technic than appears in his other article.)

FINAL TECHNIC

The technic ultimately worked out was as follows:

Enemas and starvation were dispensed with. One hour before the injection of avertin, the dog was weighed, and 10 mg. of morphine sulphate per kilogram of body weight was injected subcutaneously. The kilogram dose of avertin having been decided on, the amount of avertin fluid to be used was calculated from the body weight. (Avertin fluid is a 100 per cent solution of avertin in amylene hydrate.) From the figure thus obtained, the amount of distilled water necessary to make a 3 per cent solution of avertin was calculated. This quantity of distilled water was heated to 40 C., and the avertin was added from a 5 cc. pipet graduated in 0.1 cc. The mixture was agitated until solution was complete, and immediately injected.

With the dog held on his side by an assistant, a 30 French soft rubber catheter was inserted from 7 to 10 inches into the rectum. To the free end of the tube was attached a glass syringe with a capacity of about 30 cc., and with a tapering nozzle which fitted snugly into the tube. This syringe was used as a funnel; the rubber bulb was used merely to start the flow and to overcome any mechanical blockage, such as feces, in the orifice of the tube; the fluid was allowed to run in by gravity, with the syringe held not more than a foot higher than the anus.

If only avertin was being given, the tube was withdrawn as soon as all the fluid had been injected; if ether was to be injected, the tube was left in place and clamped, and after an interval of from ten to twenty minutes, the syringe was filled with oil-ether, the clamp removed, the injection completed, and the tube withdrawn. In one case, when anesthesia of long duration was required, the tube was left in place after injection of the first dose of ether; four hours later, when the dog began to move, a second dose of ether was injected, with prompt deepening of the anesthesia.

The ether solution used for these experiments was made up fresh for each dog; the calculated amount of ether was measured out, and half that amount of albolene was added. The mixture was not heated, but was shaken to insure thorough mixing, and injected at room temperature.

The addition of morphine to the technic obviated what had previously been the most serious difficulty, namely, forcible expulsion of the oil-ether mixture; but with the larger quantities of solutions injected there was frequently a leakage through the relaxed anus. This could be fairly well controlled by pressure on a folded gauze pad placed over the anus; if a tendency to leakage persisted more than a few minutes, pressure was continued by leaving the pad in place, pulling the tail forcibly forward between the legs, and clamping the hair of the tip of the tail to the hair of the abdomen.

Anesthesia was considered to have begun as soon as the dogs fell asleep, usually from three to five minutes after the injection of avertin. It is true that surgical anesthesia does not begin as soon as a dog falls asleep, but only after an interval of from ten to twenty minutes of deepening narcosis; as stimulation during this period is known to hinder and delay the development of deep narcosis, it was assumed that less error would be introduced by taking the time when the dogs fell asleep than by determining the actual onset of surgical anesthesia by repeated painful stimuli.

Anesthesia was considered to continue until application of a Kocher clamp to the skin of the flank evoked a definite response. The first response was usually a lifting and turning of the head toward the clamped flank. The movement was

uncertain and slow, and the effort was usually abandoned after a second or two. Occasionally, dogs began to move spontaneously before they responded to the clamp; in these cases, the time that movement began was considered to mark the end of anesthesia. In actual operation, supplemental anesthesia would not be required for some time after this point, as the dogs remained stuporous for a considerable period, particularly after the larger doses, and in a few instances relapsed into complete anesthesia from which painful stimuli did not arouse them.

Comment on Technic.—Adoption of this technic resulted in immediate success. Ether was retained almost invariably (expelled twice in twenty-six experiments), and increasing doses up to a maximum of 9 cc. per kilogram were successfully given.

An interesting question raised by the use of this large dose of morphine was whether in the presence of such a stupefying dose any subsequent procedures could lead to valid conclusions.

One fact pointing toward the affirmative answer was that an hour after injection of morphine, the dogs, though decidedly sleepy, could easily be aroused, and seemed only slightly more narcotized than many human patients who have had ordinary preoperative medication.

I took as the final criterion the pragmatic test, and found that, except in the higher ranges, increases in dosage of avertin or ether, or both, caused fairly constant increases in the duration of anesthesia.

Beckman, using oil-ether rectal anesthesia in dogs, with and without morphine, also concluded that morphine in this dosage does not invalidate the results.

In connection with the injection of morphine, it is of interest that while salivation was fairly common, vomiting or defecation was the exception rather than the rule, even in dogs that were receiving morphine for the first time.

Results with Small Doses.—Having overcome the difficulty caused by expulsion of the ether solution, I carried out a group of experiments using small quantities of avertin (0.1 Gm. per kilogram), followed either by no ether or by ether in doses of from 1 to 2 cc. per kilogram (see table 2).

These experiments demonstrated that addition of ether in doses of 1 cc. per kilogram produces a slightly but definitely longer period of anesthesia than avertin alone, and that a dose of 2 cc. of ether per kilogram causes a marked prolongation. Five dogs received this dose, i. e., avertin, 0.1 Gm. per kilogram, followed by ether, 2 cc. per kilogram. The minimum length of anesthesia obtained was one and three-quarter hours; the maximum three and one-half. It is of interest that the dog which remained anesthetized for three and one-half hours was subjected to a gastric resection and bilateral vagotomy, the operation lasting two and three-quarters hours. During the last hour of the operation, the dog made occasional slight movements, but supplemental anesthesia was not required, and ten minutes after the close of the operation he did not respond to a Kocher clamp applied to the flank.

Margin of Safety.—After this combination rectal anesthetic had been demonstrated to produce in dogs a measurably longer period of anesthesia than avertin alone, determination of the margin of safety

was next in order. For this purpose I took three dogs which had been used in previous experiments, one of them many times, and gave to one increasing doses of avertin alone; to the second, increasing doses of ether with a constant preliminary dose of avertin, and to the third, increasing doses of both avertin and ether. The results obtained are shown in table 2.

Lethal Dose.—There were no fatalities. The nearest approach to a lethal dose was avertin 0.9 Gm. per kilogram. In this case the dog was deeply anesthetized at midnight, thirteen hours after the administration of avertin, with greatly depressed respiratory and cardiac rates (respiration, 10; heart rate, from 42 to 46 per minute). At 8 a. m.

TABLE 2.—*Experiments on Dogs with Morphine (10 Mg. per Kilogram), with Avertin and with Avertin-Ether*

Avertin in Gm. per Kg.	Ether in Cc. per Kilogram									
	0	1	2	3	4	5	6	7	8	9
0.1	½ ½ 1½ 1¼	½ ¾ 1¾ 2	1¾ 2 2½ 3 3½	2 2½	3 4½	4 4½ 6 (divided dose)	..	..	3½	7
0.2	2¼	..	..	..	3	..	..	..	..	..
0.3	2	..	..	..	..	5	..	..	..	..
0.4	3¼	..	..	..	..	..	6½	..	..	..
0.5	4 6 7½	..	..	..	..	6	..	4½	..	..
0.6	4½	..	..	..	..	..	..	..	5	..
0.7	4½ 5½	..	..	..	..	..	..	..	..	..
0.8	5½	..	..	..	..	..	..	..	..	..
0.9	13-20 (see text)	..	..	..	..	..	..	..	..	..

Figures represent duration of anesthesia in hours.

the next day he was rolling around the cage in a groggy and uncoordinated manner. This depressed mental and physical condition persisted all day, but on the following morning he was alert and active, and seemed completely recovered. The dogs which received avertin plus ether showed no alarming respiratory or cardiac depression, and when the experiments were stopped because the volume tolerance of the dogs' rectums had just about been reached, the doses seemed to be well below the lethal amounts.

Ether in Divided Doses.—In one instance, when anesthesia was required for a long blood-clotting experiment, I gave ether in divided doses, with good results. The dog was anesthetized with avertin, 0.1 Gm. per kilogram, plus ether, 3 cc. per kilogram. At the end of four hours, the anesthesia was beginning to wear off, but injection of an

additional 2 cc. of ether per kilogram caused prompt deepening of the anesthesia, which lasted for two hours more.

Comment on Results.—The inconstancy of the results with very large doses may be accounted for in two ways. In the first place, the actual quantities of fluid required are tremendous, compared to the size of the dog. For instance, the dog which received avertin, 0.6 Gm. per kilogram, and ether, 8 cc. per kilogram, weighed 14.4 kilograms. Consequently he was given 4.3 cc. of morphine sulphate solution, 288 cc. of avertin solution and 173 cc. of oil-ether mixture. The magnitude of these figures (461 cc. injected rectally into a dog weighing 14.4 kilograms) may best be realized by multiplying both figures by five; that is, this is equivalent to giving a 2,300 cc. retention enema to a 72 kilogram (158.4 lb.) adult human being. In these ranges, there is naturally some leakage, even with compression of the anal region; this particular dog lost a quantity of avertin solution estimated at 15 cc. (a little over 5 per cent of the total amount injected) and about 5 cc. of the ether solution (about 3 per cent). Leakages in the other experiments were of about the same magnitude.

The more important factor, I believe, was the tolerance acquired by the dogs to morphine and, possibly, to avertin. Previous experimentors have concluded that avertin given repeatedly neither produces tolerance nor has a cumulative effect. My results agree with the latter, and the former is certainly not disproved, in view of the well known fact that tolerance to morphine is readily produced. I did not perform the critical experiment of habituating a dog to morphine and then giving him avertin.

I did, however, wish to check my results with the larger dosages on fresh dogs. Two dogs were obtained which had never been subjected to avertin, and after the usual dose of morphine, were given avertin, 0.5 Gm. per kilogram. The anesthesia obtained lasted six hours in one dog and seven and one-half in the other, as compared with four hours in the habituated dog. One of these dogs was then given avertin, 0.7 Gm. per kilogram, with anesthesia lasting only four and one-half hours, as against five and one-half hours in the habituated dog. The other was given avertin, 0.5 Gm. per kilogram plus ether 5 cc. per kilogram. Anesthesia in this case lasted six hours.

These four experiments gave definitely inconstant results, and raised the question whether the colonic distention caused by these large quantities of fluid does not introduce error by preventing absorption of a good proportion of the injected drugs. At any rate, these doses seem well within the fatal limits.

One additional factor which may have had a bearing on the lack of uniformity when different dogs were used is that larger dogs seem to be more affected by the preliminary morphine, and possibly also by the other drugs.

Typical protocols follow:

Experiment 105, 2-26-32

Dog 536	10 Kg.	Morphine, 10 mg. per Kg. Avertin, 0.1 Gm. per Kg. Ether, 5 cc. per Kg.
12:25 p. m.	Morphine injected	
2:10	Avertin injected	
2:13	Asleep	
2:27-2:30	Ether injected; 5 cc. ± lost	
3:00	Deep anesthesia	
3:30	Deep anesthesia	
3:50	Deep anesthesia	
4:10	Deep anesthesia	
4:30	Deep anesthesia	
5:00	Deep anesthesia; drawing up of hind legs with each inspiration	
5:20	Same	
5:35	Same	
5:55	Same	
6:10	Same	
6:25	Same	
6:35	Rouses at noise; slow response to Kocher	

Anesthesia for 4⅓ hours

Experiment 112, 3-11-32

Dog 447	13.8 Kg.	Morphine, 10 mg. per Kg. Avertin, 0.4 Gm. per Kg. Ether, 6 cc. per Kg.
10:35 a. m.	Morphine injected	
12:12-12:15	Avertin injected	
12:15	Asleep	
12:23-12:31	Ether injected; 10 cc. ± lost	
12:25	Deep anesthesia	
2:00	Deep anesthesia	
3:05	Deep anesthesia	
4:15	Deep anesthesia	
5:00	Deep anesthesia	
5:35	Deep anesthesia	
6:00	Deep anesthesia	
6:20	Deep anesthesia	
6:30	Deep anesthesia	
6:50	Slow and feeble response to Kocher	

Anesthesia for 6½ hours

Autopsy.—The three dogs which had been used for the increasing dosage experiments were killed, and autopsies were performed. One of these animals had had twelve injections of avertin without morphine and twelve injections with morphine in the course of eleven months. The other two dogs had been used fourteen and ten times, respectively.

In no case were gross lesions found in the liver, kidneys or rectum. Microscopic sections were made of these organs in all three dogs. I could find no histologic lesions in any section, nor could Dr. A. Purdy Stout of the department of surgical pathology.

COMBINATION ANESTHESIA IN OPERATIONS ON THE DOG

Combination avertin-ether rectal anesthesia can be used to advantage in certain surgical procedures on the dog, particularly for long operations or experiments. I used it successfully for two abdominal operations, one bronchoscopy and one long experiment in blood-clotting. Avertin alone (from 0.1 to 0.2 Gm. per kilogram), with preliminary morphine medication, is undoubtedly sufficient for bronchoscopies and other procedures requiring comparable depth and duration of anesthesia.

The advantages of avertin-ether rectal anesthesia are: (1) the length of anesthesia induced by small doses, (2) the fact that no inhalation anesthesia is necessary and (3) the typically smooth course of the anesthesia, with no struggling and little or no salivation.

The dose recommended for routine administration is avertin 0.1 Gm. per kilogram, followed by ether, from 2 to 3 cc. per kilogram, given according to the technic outlined.

Disadvantages of this anesthesia in dogs are: 1. The dog must be weighed and morphine injected from one and one-fourth to one and one-half hours before the operation is to be begun. 2. The anesthetic solutions must be made up carefully and injected from twenty to thirty minutes before the operation. 3. Morphine and, particularly, avertin are expensive drugs. While these factors will preclude routine use of this anesthesia in most laboratories, I feel that it has a field of definite usefulness.

There were two failures in giving this anesthesia for operations. In one case, the dog had colitis due to distemper, which I should have considered a definite contraindication, except that the distemper in his respiratory tract made inhalation anesthesia seem even less feasible. In this dog, the usual doses produced no anesthesia at all. In the other dog, anesthesia was successful until the operator exposed and manipulated the nerves in the region of the knee joint. The anesthesia was not deep enough for this procedure, and ether inhalation had to be instituted.

COMBINATION ANESTHESIA IN SURGICAL PROCEDURE ON MAN

I have had no clinical experience with this combination. York [5] has used it in over one hundred cases, including a number of thoracoplasties, and reports in a personal communication a lower mortality than he would expect in a similar group of cases with other anesthetic agents. He gives the usual dose of avertin (from 60 to 100 mg. per kilogram), and follows this with from 30 to 60 cc. (1 to 2 ounces) of ether. He

5. York, Arthur J.: Personal communication.

has also given ether in divided doses with success. Further cautious clinical trial seems warranted, in view of the experience of York and Schork and of the experimental work here presented.

SUMMARY

In albino rats, rectal injections of avertin followed by oil-ether gave a definitely longer period of anesthesia than avertin alone.

Similar procedures were carried out in dogs, and a successful technic evolved. As in rats, the combination of avertin and ether gave a longer period of anesthesia than avertin alone.

The margin of safety seemed to be wide. No dog died, in spite of doses at least three times as large as the effective anesthetic dose, which is considered to be avertin, 0.1 Gm. per kilogram, followed by ether, from 2 to 3 cc. per kilogram.

Autopsies on three dogs which had been subjected to many anesthesias failed to reveal any gross or microscopic pathologic condition in the kidneys, liver or rectum.

This combination anesthesia is recommended for use in surgical procedure on dogs in selected cases. Its use in man, as advocated by York and Schork, is considered.

CONCLUSIONS

1. In the dog, combination avertin-ether rectal anesthesia gives a definitely longer period of anesthesia than avertin alone; the procedure is safe; the lethal dose is at least three times the effective anesthetic dose. The clinical experience of York and Schork is thus corroborated experimentally.

2. On the basis of both clinical and experimental experience a wider clinical use of this combination anesthesia seems advisable.

FIFTY-FIRST REPORT OF PROGRESS IN ORTHOPEDIC SURGERY

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(Concluded from page 816)

OSTEOMYELITIS

Acute Osteomyelitis in Children.—Statistics from eight British hospitals supported Wakeley's [25] contention that acute osteomyelitis was a disappearing disease. He suggested that the decrease of incidence was due to better hygiene and the earlier removal of possible septic foci. He questioned the general belief that trauma played an important part in the causation of the disease. Early drainage was indicated in all cases, a trephine being used at first in the juxta-epiphyseal diaphysis. The efficacy of multiple drill holes as a means of instituting drainage was doubted. In late cases, in which the whole of the diaphysis was involved in the disease, diaphysectomy followed by bone grafting was advised. Fixation of the limb was considered to be necessary to rest the inflamed bone and eliminate pain.

CHRONIC ARTHRITIS

Treatment of Chronic Arthritis of the Spine.—Swaim and Kuhns [26] discussed the treatment of atrophic arthritis of the spine. Five essentials of treatment were considered: (1) early diagnosis, (2) arrest of the general disease as quickly as possible, (3) improvement in the motion of the ribs, (4) correction of the forward droop of the spine and (5) improvement of the patient's general health.

25. Wakeley, C. P. G.: Brit. M. J. **2**:752, 1932.

26. Swaim, L. T., and Kuhns, J. G.: Arch. Phys. Therapy **13**:517, 1932.

Permanent cure came only slowly. The rapid improvement sometimes seen with the use of drugs or vaccines was usually not permanent. Important measures, such as proper diet, intestinal regulation, endocrine therapy (occasionally) and correction of faulty body mechanics, needed to be followed for many months to have a permanent beneficial effect. Emphasis was placed on the importance of increasing vital capacity and the respiratory expansion of the ribs. Without this expansion of the ribs, death from pneumonia was frequent. Rest was an important factor in conquering the disease. Restoration of the normal curves of the back and improvement in general body mechanics was brought about by exercises, and a gradual correction of the forward flexion of the spine by means of supporting plaster shells and bed positions. No rapid corrections or manipulations were attempted. Improvement in the position of the spine and thorax was accompanied by improvement in the patient's general health and resistance. Patients were kept in bed until all pain and muscle spasms had subsided. Gentleness was the keynote of all therapeutic exercises. Involvement of the hips presented the serious and intractable features of the disease. Once the arthritis had become firmly established in the hip, it usually went on to ankylosis, and little could be done except to prevent deformity.

CIRCULATORY DISTURBANCES OF THE EXTREMITIES

General Care of Peripheral Vascular Disease.—Reid [27] stressed certain practical points in the care of peripheral vascular disease and emphasized particularly the importance of intelligent cooperation on the part of the patient in carrying out treatment of these conditions. The position of maximum circulation for the affected part should be determined in each case. Too much elevation often encouraged the development of gangrene. The necessity of keeping the skin soft and the extremity warm, and the avoidance of trauma were mentioned. The Buerger exercises carried out consistently and the maintenance of an adequate fluid intake were recommended. The author advised doing amputations between two tourniquets in cases of infected or gangrenous extremities.

[ED. NOTE.—The article is well written and practical; while containing nothing particularly new, it does summarize well the important points in the care of these patients. It should be read by all physicians interested in surgery of the extremities.]

Quantitative Determination of Vasoconstrictor Spasm.—Morton and Scott [28] emphasized that before a decision is made as to the type of

27. Reid, M. B.: Ann. Surg. **96**:733, 1932.

28. Morton, J. J., and Scott, W. J. M.: Ann. Surg. **96**:754, 1932.

treatment to be applied in a particular case of peripheral vascular disease, it is first necessary to determine whether the complaint is due to a failure of the blood supply, and secondarily whether this failure is due to organic occlusion or to sympathetic vasoconstriction or to a combination of these. Observations were made on normal persons and on persons suffering from vascular disease to determine the vasodilatation level. They found the most convenient method to be the conduction nerve block. In selected cases, lumbar or cervical sympathectomy was considered advisable, if by the nerve block test it could be established that vasodilatation could be produced, the surface temperature raised and spasm relieved. The establishment of collateral circulation was considered an important result of sympathectomy.

Sympathetic Ganglionectomy and Trunk Resection in Circulatory Disturbances.—Mayo and Adson [29] wrote extensively concerning the surgery of the sympathetic nervous system, and reviewed the anatomy, historical facts and the surgical technic of the operative procedures. The operation of sympathetic ganglionectomy as applied to Raynaud's disease, thrombo-angiitis obliterans and scleroderma was presented, and the results in a large series of cases recorded.

[ED. NOTE.—This article gives an excellent summary of the operative procedure and its clinical applications.]

BONE AND JOINT SURGERY

Whitman's Reconstruction Operation on the Hip Joint.—Lowendorf [30] based his remarks on a study of the end-results in 15 of 18 cases in which the Whitman reconstruction operation had been done. The indications for the operation were: (1) ununited fracture of the femoral neck, (2) old slipped epiphysis, (3) pathologic dislocation of the hip and (4) osteo-arthritis or traumatic arthritis of the hip. The ideal result obtainable was a movable, painless and stable hip. Good results were obtained in 11 of the 15 cases. The operation was done in 8 of these for nonunion of the femoral neck. Good results were obtained in all cases of this type. Operation was performed in the other cases for arthritis. The conditions in the 4 cases in which poor results were obtained were: osteo-arthritis, old suppurative arthritis, generalized arthritis and Charcot's joint. In 14 cases, the average flexion contracture was 14 degrees and the range of flexion 67 degrees, approximately 20 degrees abduction and 20 degrees adduction. Stable hips were obtained in 14 of the 15 cases. Pain was relieved in 12 cases.

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29. Mayo, W. J., and Adson, A. W., Ann. Surg. **96**:733, 1932.

30. Lowendorf, C. S.: Am. J. Surg. **18**:64, 1932.

MISCELLANEOUS

Cerebral Injuries at Birth.—Phelps [31] advised a careful study of cerebral injury at birth from the social, mental and physical standpoint, before outlining any form of treatment. For the study of the physical side, he found motion pictures of great value. He discussed "dyskinesia" under the headings of (1) spasticity, (2) athetosis, (3) overflow of "synkinesia," (4) incoordination or ataxia and (5) tremor. The principles of treatment were: (1) relaxation, which was very essential before any progress could be made, (2) strengthening exercises and (3) reciprocal motions, to be gained only after relaxation had been accomplished. (4) Active stretching exercises were useless. (5) Surgical intervention should be considered only after the aforementioned preliminary forms of treatment had been tried. The various operative procedures, such as tendon division and nerve resections, were discussed.

Angle of Gait.—Morton [32] examined the angle of gait of 147 natives of Central Africa (87 men, 30 women and 30 children), and found that the natural angle of gait was 7.5 degrees of out-toeing. This finding corresponds closely with those of Dougan and Patek, who made similar studies on college men and women, but is contrary to the ideas of some orthopedic surgeons who teach patients that toeing in or walking with feet directly forward is the ideal. The author analyzed the benefits of two functional elements in the mechanics of stride: (1) lateral stability effected by widening the position of the feet, i. e., toeing out, and (2) a forward thrust from behind the moving center of body weight brought about by having the heels placed more closely together.

Glycine Treatment for Progressive Muscular Dystrophy.—The creatine-creatinine excretion and the tolerance to creatine were studied by Milhorat [33] in cases of muscular dystrophy. He observed that the feeding of glycine with or without phosphate raised the elimination of creatine in 14 cases of progressive muscular dystrophy. No increase in the elimination of creatine was observed in secondary muscular atrophies. Consequently, the feeding of glycine served as a differential diagnostic measure between primary and secondary degeneration. Under this treatment, the author found rapid improvement in the muscles not seriously atrophied with later less rapid progress. Histologic evidence of muscular tissue from 3 patients was difficult to interpret. Muscular fibers were either quite atrophied or entirely normal. No middle stages were observed.

[ED. NOTE.—An easily available inexpensive source of glycine is gelatin.]

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31. Phelps, W. M.: J. Bone & Joint Surg. **14**:773, 1932.
32. Morton, D. J.: J. Bone & Joint Surg. **14**:741, 1932.
33. Milhorat, A. T.: Deutsches Arch. f. klin. Med. **174**:487, 1933.

Ruptures and Tears of Muscles of the Lower Extremity.—Fifteen cases of ruptured muscles in the lower extremity were reported by Gilcreest.[34] There was a history of trauma in all the cases. In only 2 was the trauma trivial enough to cause comment: A woman of 35 ruptured the quadriceps femoris tendon while bending over with the knees flexed. Another patient ruptured the plantaris when stepping off a street car. Ruptures of the vastus internus quadriceps tendon, rectus femoris, patellar ligament, semitendinosus, inner head of the gastrocnemius, achilles tendon and plantaris were reported. The author concluded that partial or complete ruptures of the muscles and tendons of the leg occurred more often than was generally supposed. Articular symptoms, particularly in the knee, might mislead one in making a diagnosis. Early surgical repair gave good results. The prognosis was good if proper treatment was given early.

Pneumoroentgenogram of the Knee Joint.—With the aid of a pneumothorax apparatus, Schum[35] was able to inject from 60 to 200 cc. of unfiltered air into the knee joint. The joint puncture was usually made under local anesthesia, lateral to the patella, and air was not injected until the operator was sure that the needle was free in the joint. To test this, if there was any doubt, some salt solution was first run through. This was done to avoid air emboli, the major danger in this procedure. The resultant roentgenograms, taken stereoscopically, anteroposteriorly and laterally, were most interesting and often of much clinical value. Especially interesting were pictures taken in lesions of the lateral ligaments. Lesions of the menisci often were recognizable, but findings were used only when positive. In 54 cases clinical and roentgenographic findings pointed to injuries of the meniscus; in 88.7 per cent of these the conditions were found to have been correctly diagnosed when checked at operation. Of the remaining 11 per cent (6 cases), the diagnosis in 3 cases was indicated in any event by the demonstrable presence of free bodies and injuries to the fat pad, while in 3 cases pneumoroentgenography definitely failed to be of value. No trouble was encountered in performing the injection of air 114 times on 98 patients. A sense of fulness was usually experienced when the joint was properly filled, but no general symptoms appeared. The needle was left in place during the taking of the roentgenograms to avoid subcutaneous emphysema. After roentgen exposures were completed, the air was gently massaged out. Some clinical improvement was noted in some cases simply as a result of the distention.

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34. Gilcreest, E. L.: Ruptures and Tears of Muscles and Tendons of Lower Extremity: Report of Fifteen Cases, J. A. M. A. **100**:153 (Jan. 21) 1933.

35. Schum, H.: Deutsche Ztschr. f. Chir. **238**:1, 1933.

Osteochondritis Necroticans "Findens" (Divided) of the Sesamoid Bone of the First Metatarsal.—Kimmelstiel and his co-workers[36] presented a careful study of 35 cases of this not unusual syndrome. Roentgenograms and sections revealed changes similar to those seen in osteochondritides. Clinically, pain under the joint of the big toe of varied duration and intensity was the outstanding complaint. Only seldom was the onset acute. In only 3 cases was sudden trauma an apparently definite factor. On examination, local tenderness and pain on hyperextension of the toe were marked. Occasionally swelling existed. Static deformities of the feet were often found. The roentgen findings were most significant: disintegration, changes in form, vacuolation, sequestrum formation and loss of normal trabecular structure might be seen. Frequently portions of a sesamoid up to one-third its volume were seen as free bodies. Conservative pad support and physical therapy gave relief in most of these cases. Roentgen findings in one case did not appreciably change in one and one-half years. To demonstrate the sesamoid changes best, the authors placed the patient in a prone position. The big toe was strongly dorsiflexed, placed on the film, and the x-rays were projected from the dorsal aspect. The interpretation of the pathologic process in terms of causation was difficult, but there was evidence suggesting the effect of gradual, repeated traumas as the probable cause. In one specimen, arthritis of the joint was present as well. Removal of the affected sesamoid was easily accomplished. When conservative therapy was not successful, removal of the sesamoid through a plantar incision was indicated and led to rapid cure. No ill effects were noted in consequence of its excision.

[Ed. Note.—Pain referable to the sesamoid is rare, and excision of the sesamoid is rarely justifiable in the experience of the editors.]

Study of the Blood Supply of the Ligamentum Teres and Its Relation to Circulation of the Head of the Femur.—Chandler and Kreuschner[37] studied 114 round ligaments from 68 cadavers, the average age of which was 48 years. In all but one of the ligaments they found blood vessels which branched in the femoral head. In any surgical operation on the hip, preservation of the intact round ligament was considered most important.

Circulation of the Head and Neck of the Femur.—Forty-one cases of intracapsular fracture of the neck of the femur were studied by

36. Kimmelstiel, P.; Kremger, K., and Richter, H.: Arch. f. klin. Chir. **172:** 403, 1932.

37. Chandler, S. B., and Kreuschner, P. H.: J. Bone & Joint Surg. **14:**834, 1932.

Wolcott.[38] The cases with nonunion showed increased density of the head by roentgen examination. In addition early (time intervals were not given) absorption of the femoral neck was noted. This continued until the entire neck had disappeared. Four patients were operated on, and the ligamentum teres removed for examination. In all cases microscopic sections showed patent blood vessels in the ligamentum teres. The ages of the patients were 60, 72, 76 and 63. Blood supply through the ligamentum teres was therefore considered to be a definite factor in maintaining the nourishment of the femoral head. Consequently, operations on the hip joint should be designed and carried out in such a way as not to disturb or injure the vessels coming through the ligamentum teres. The author was careful to state that the femoral head had other sources of nourishment and that other factors besides circulation entered into the production of nonunion.

Production of Increased Compression Strength of Bone.—Using a special machine which measured the compression strength of bone, Ross [39] showed that the operations carried out by Barth and Archibald to increase the strength of bone in cases of juvenile osteomalacia and fragilitas ossium were not effective. The operations consisted of removing subperiosteally longitudinal segments from the long bones, grinding or rongeuring these and placing them in the marrow cavity. Ross showed, however, that if detached strips of muscle were placed in the marrow cavity, deep to the fragmented bone, that the strength of the healed bone was enormously increased, in 1 case over 100 per cent. Histologically, the muscle was found to have been replaced by bone, with a consequent final thickening of the cortex. At least four months were required for the development of the extra strength.

Calcareous Deposits in the Supraspinatus Tendon.—Seven cases in which operation was done for calcareous deposits in the supraspinatus tendon were described by Emslie.[40] In three of these, small cavities were found in the head of the humerus. Examination of 162 mg. of the ash of one of the deposits demonstrated the composition to be 116 mg. of calcium phosphate, 15 mg. of calcium carbonate and 31 mg. of undefined ash. All cultures taken at operation were sterile. The most characteristic finding was tenderness over the tip of the shoulder. Total extirpation, with curetting of the cavity, if present, was advocated, even if this entailed the opening of the shoulder joint. The approach was by retracting the anterior border of the deltoid muscle.

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38. Wolcott, W. E.: Circulation of the Head and Neck of the Femur: Its Relation to Nonunion in Fractures of the Femoral Neck, J. A. M. A. **100**:27 (Jan. 7) 1933.

39. Ross, D.: Brit. J. Surg. **20**:337, 1932.

40. Emslie, R. C.: Brit. J. Surg. **20**:190, 1932.

Antigrowth Principle Derived from Parathyroid Extract.—By injecting an extract of parathyroid gland from which the blood calcium-raising principle has been removed into various experimental animals, Robinson and Thompson [41] showed that a great retardation of growth was brought about. The retardation was approximately equivalent to the quantity of extract given. In some cases tolerance was developed. The antigrowth principle was not isolated, but some information was obtained relating to its properties.

FRACTURES

Colles' Fracture.—Three years of experience in treating Colles' fractures with plaster-of-paris fixation for five weeks, as advocated by Böhler, convinced Platt [42] that nothing was lost in either the function or the nutrition of the part by the elimination of physical therapy, and that the risks of recurrent deformity were minimized.

[ED. NOTE.—This is an interesting point of view. The editors believe that physical therapy still has a place in the treatment of Colles' fracture.]

Treatment of Fractures in the Light of Their Ischemic Complications.—Girdlestone [43] discussed ischemic complications of fractures and divided them as follows: (1) Volkmann's ischemic contracture, the result of sudden localized pressure; (2) an ischemia due to relatively prolonged and widespread contraction and disuse, occurring characteristically in older patients and due to a localized sympathetic dysfunction, and characterized by swelling, coolness and stiffness of the part; (3) ischemia at the site of a fracture, as in the case of the carpal scaphoid, the femoral neck and sometimes the long bone, designated by the author as traumatic arterial ischemia. The treatment of each was discussed, i.e.: (1) early recognition of a Volkmann's contracture, and fascial decompression if necessary; (2) avoidance of too prolonged fixation, and (3) accurate reduction and efficient fixation followed by proper forms of physical therapy to improve circulation to the part, and improvement of the local sympathetic response.

RESEARCH

Studies on Tendon Repair.—Mason and Shearon,[44] in an exhaustive experimental study of tendon repair in dogs, found that within one

41. Robinson, M. H. B., and Thompson, J. H.: J. Physiol. **76**:303, 1932.

42. Platt, H.: Brit. M. J. **2**:288, 1932.

43. Girdlestone, G. R.: J. Bone & Joint Surg. **14**:755, 1932.

44. Mason, M. L., and Shearon, C. G.: Progress of Tendon Repair: Experimental Study of Tendon Suture and Tendon Graft, Arch. Surg. **25**:615 (Oct.) 1932.

week after suture of the tendon a fairly firm union was affected by proliferation of sheath tissues. This was gradually invaded and replaced by proliferation of the tendon cells themselves, a minimum of two weeks being required to establish firm union. They concluded that in suture of the tendon the sheath should be carefully closed, if possible. Accurate end-to-end approximation of the ends of the tendon was important, and defects should be bridged by free tendon grafts with their sheath tissue intact rather than by fascia, which tended to stretch. Movements might be begun on the fifth or sixth day, but no strain should be placed on the tendon before the third week.

Repair of Articular Cartilage.—In the joints of normal adult dogs, Bennett and Bauer [45] produced defects in the articular cartilage; in some they displaced the patellae so as to cause abnormal friction. They also studied the effect of disarticulation of the joint on the cartilage. The joints were studied at intervals of four, twelve, twenty-two and twenty-eight weeks. Their conclusions were: 1. Cartilage had a limited ability to repair aseptic lesions within its substance by independent regeneration of cartilage, and the repair was more perfect over the mesial femoral condyle than in the patellar groove. 2. Portions of cartilage excised from the joint and later returned to the joint cavity were usually walled off by fibrous tissue and removed from the joint space. 3. In the cases in which defects extended into subchondral bone, and in the joints in which the patellae had been displaced, the reparative changes passed through stages of fibrous tissue and fibrocartilage to the formation of an imperfect hyaline cartilage. In disarticulated joints, extensive atrophy with pannus formation over the joint cartilage developed, demonstrating the importance of proper apposition and weight-bearing of adjoining articular surfaces.

45. Bennett, G. A., and Bauer, W.: Am. J. Path. **8**:499, 1932.

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